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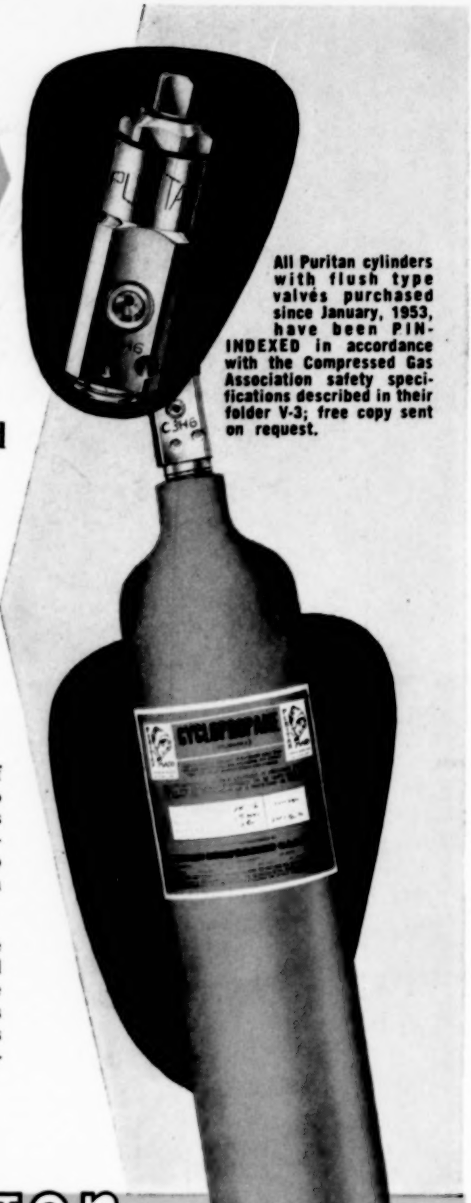

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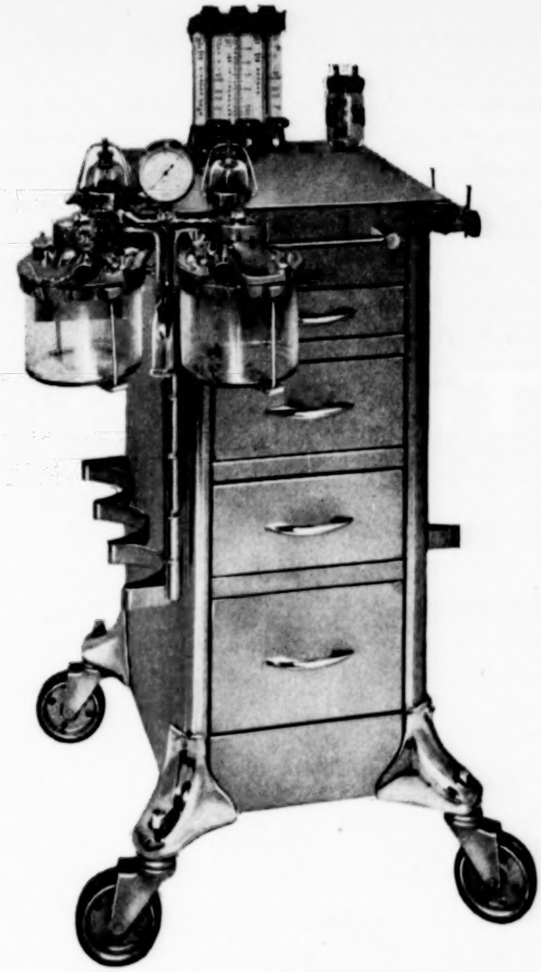

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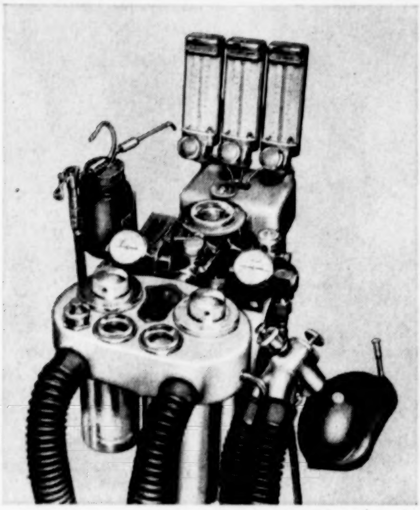

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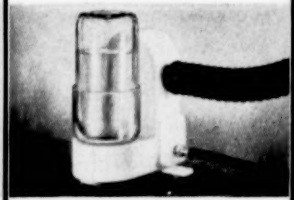

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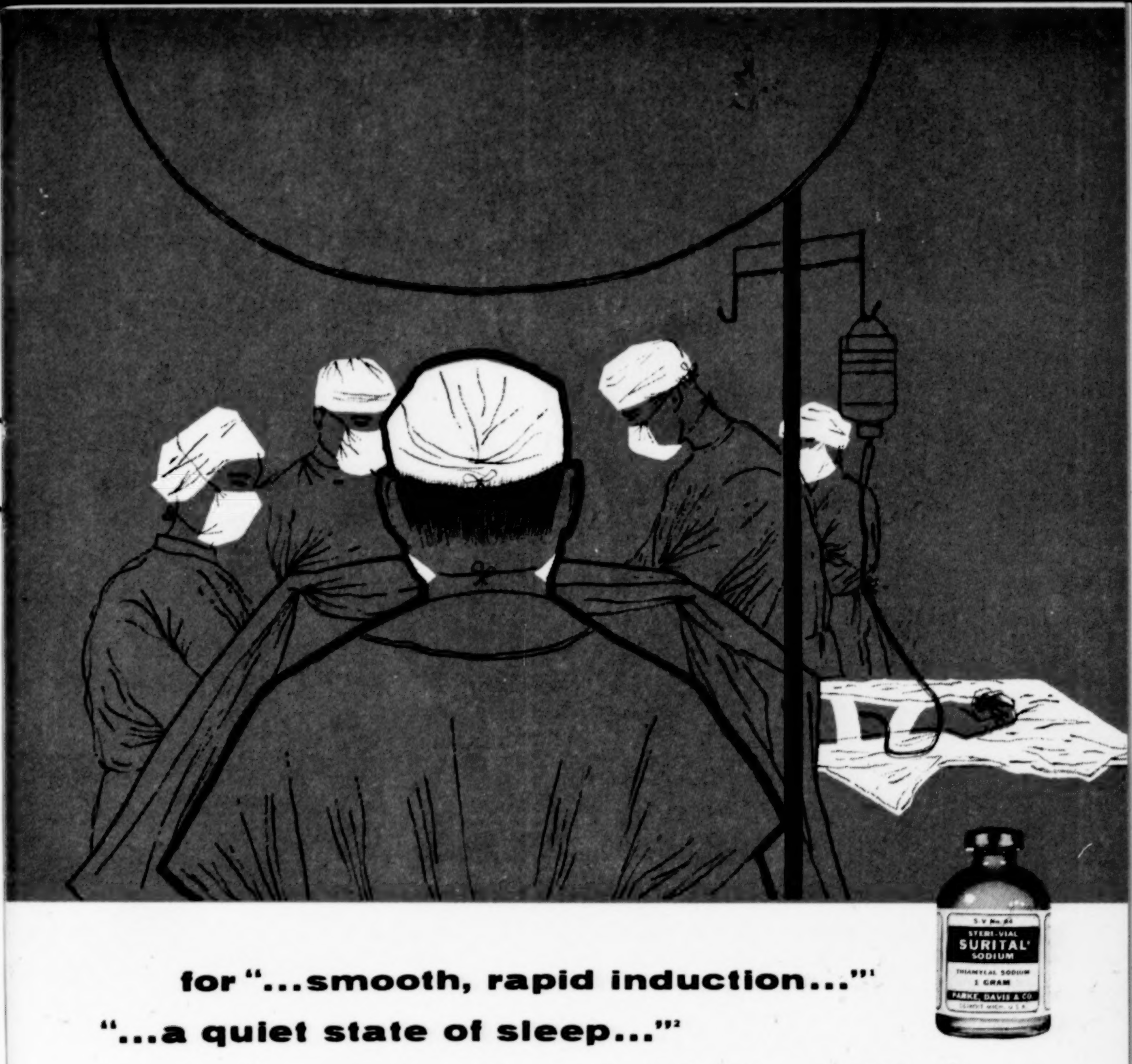
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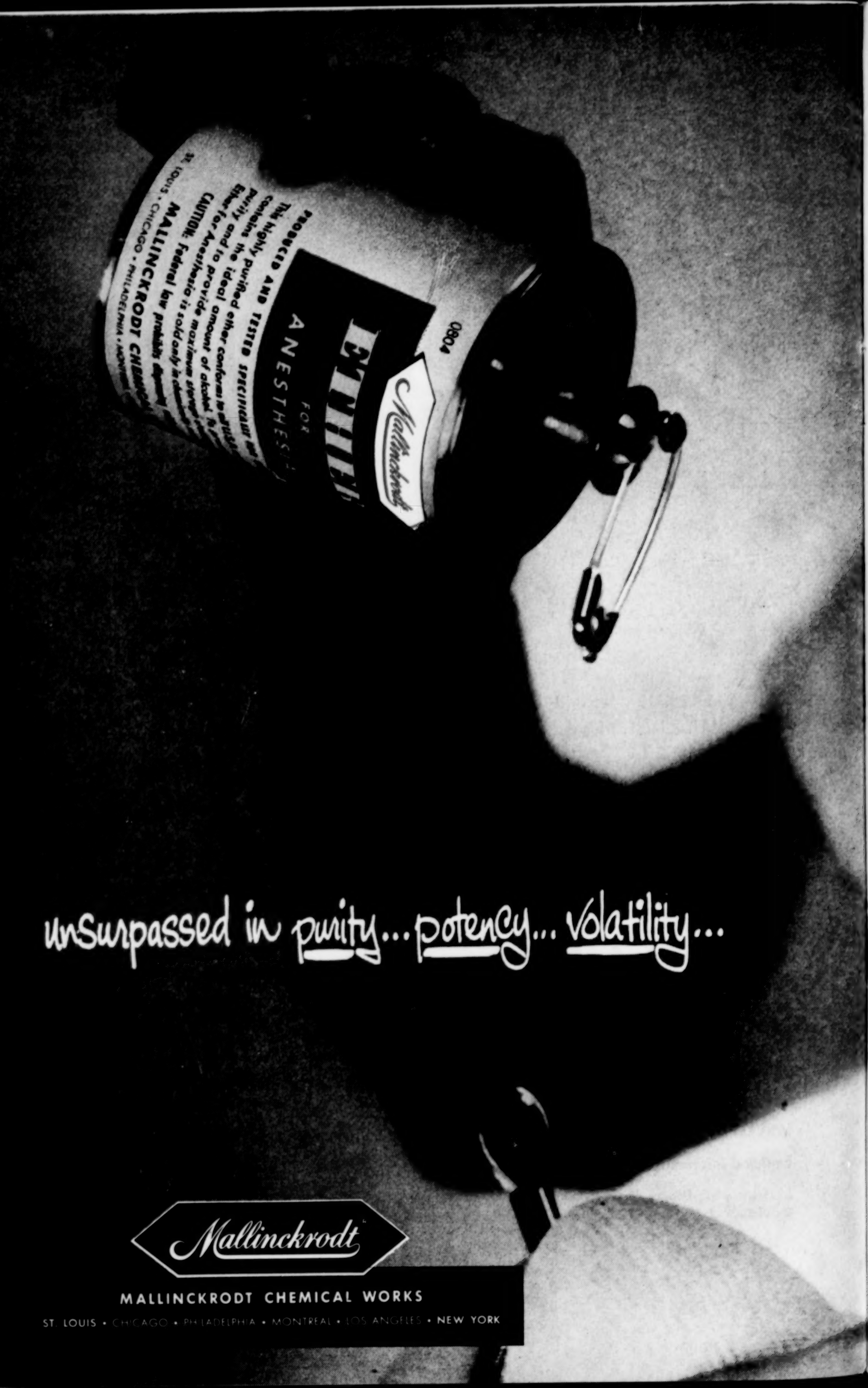
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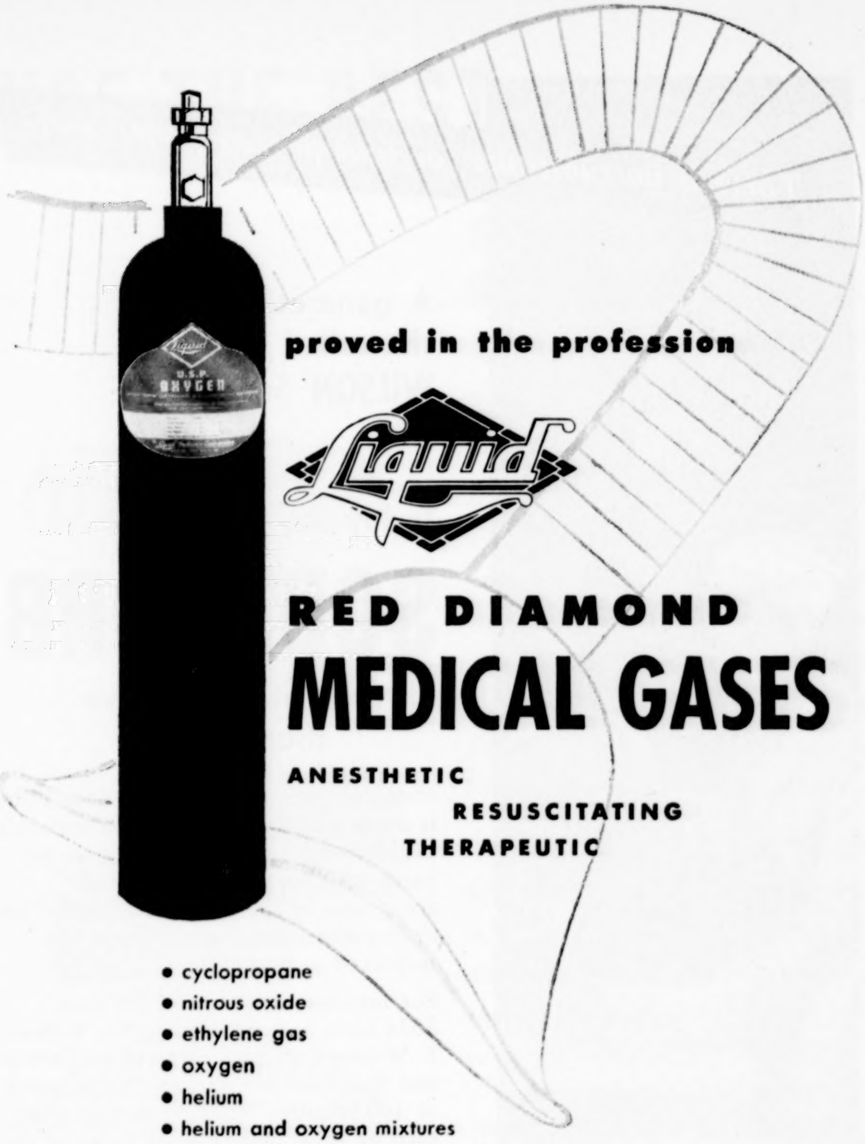

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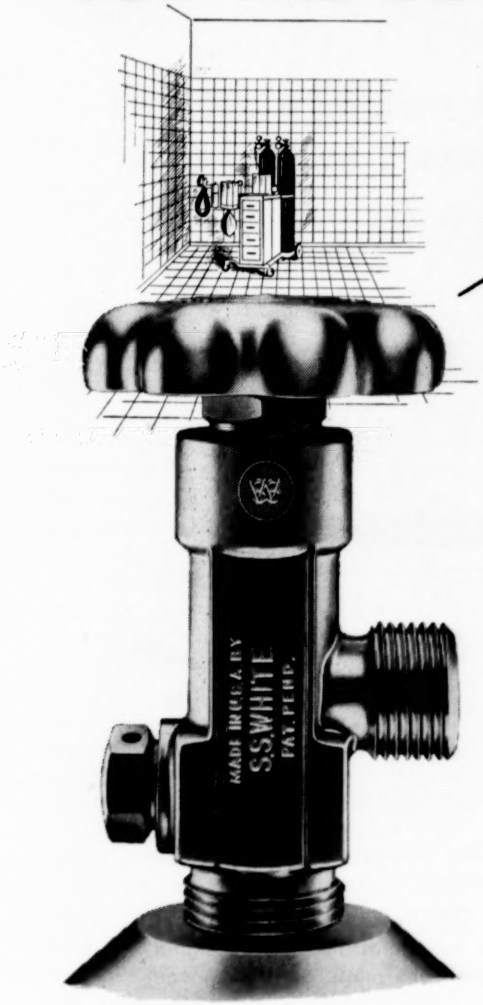

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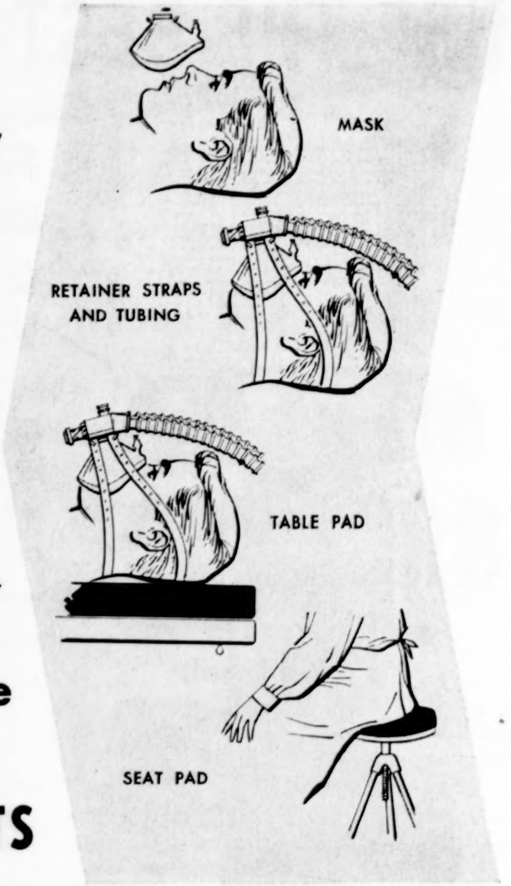

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The Role of the Anesthetist in the Control of Hemorrhage During Surgery

Exum Walker, M.D.*
Atlanta, Georgia

I must confess that I feel some reticence in presenting this paper for, as you know, I am not an anesthesiologist, but rather a neurological surgeon. I fear my remarks may reach you as all-too-familiar material and that I may be embarrassed by your scientific scrutiny. However, I am encouraged to put aside these qualms and ask you to accept me, if you will, not as one learned in anesthesiology, but as a surgeon who has had to consider some of the more practical aspects of anesthesia in carrying out certain difficult procedures.

Before going on, let me pause here to pay tribute to those of you in the field of anesthesiology whose efforts have so greatly implemented and facilitated the surgeon's task. Gone are the days when the anesthetist had as his essential function merely to render patients insensitive to pain. It is a great step forward in the field of surgery now to see the anesthetist assuming the additional responsibility of exercising control of the circulation throughout the surgical procedure and the period of recovery.

Let me continue my diversion for a moment to comment upon the cooperation between anesthetists and surgeons which has become so commonplace. Happily, we have almost forgotten the time when temperamental self-concern often threatened to supersede the patient's interest. Let us resolve to maintain our teamwork so that we may continue to anesthetize and carry out technical surgical procedures more easily and with greater safety.

In the field of surgical technic, there is probably no more important single problem than the control of hemorrhage. Indeed, only with the introduction of hemorrhage control did surgical procedures become practical at all. In general, there has been a preoccupation with local measures for controlling excessive bleeding. These have included the tourniquet, the ligature, technical speed, and more recent refinements such as the use of suction, electrocoagulation, and gelatin foam. For the routine operative case, these

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Presented at the meeting of the Southeastern Assembly of Nurse Anesthetists, April 7, 1954, Atlanta, Georgia.

*133 Doctors Building, Atlanta, Georgia.

technics appear to be adequate. Certain operations, however, such as those for aneurysms, highly vascular tumors, and vascular malformations have presented a need for some additional measures to control hemorrhage, and, as a means of performing such operations with a higher degree of safety, certain methods have been developed which have come to be known as "hypotensive technics."

These technics have as their basic principle the fact that the rate of hemorrhage from an open vessel is proportional to the intravascular pressure. Therefore, bleeding can be diminished or curtailed simply by lowering the intravascular pressure. The objective, obviously, is to lower the intravascular pressure sufficiently to diminish bleeding effectively, but not enough to impair vital circulation. This can be accomplished in a number of ways, all of which require that the anesthetist work closely with the surgeon and share the responsibility for careful planning and skillful execution.

It has been learned that patients can be carried for a limited time with the blood pressure remarkably lowered, provided there is peripheral vasodilatation, adequate oxygenation, and a sustained blood volume. This provides a bloodless, or near bloodless, surgical field which may allow the accomplishment of a surgical task that otherwise might be hazardous or impossible. Profound or prolonged hypotension should be used only when the procedure is urgently necessary, and then, only after carefully balancing the risks against the gains. In addition, both the surgeon and the anesthetist must be fully informed regarding the technic and must exercise sound judgment in its use, for there may be considerable risk to both function and life if the circulation falls short of the minimal basal level. In short, when used with caution, controlled hypotension provides a means of greatly diminishing blood loss, cutting down on operating time, and, on occasion, enabling the surgeon to remove a vascular tumor or to control serious hemorrhage which otherwise might be very difficult, if not impossible.

There is also a place for a more conservative lowering of blood pressure to minimize blood loss in a patient who bleeds or oozes excessively. It is surprising how effectively excessive bleeding stops when the pressure is lowered to 100-110 systolic in a normotensive subject or when the pressure of a hypertensive individual is lowered 30-40 mm. Some British and Swedish surgeons have used hypotensive technics routinely in all types of operations and claim that, not only is surgery greatly facilitated, but also the need for transfusion is almost negated. While such extensive use may be questioned, these experiences serve to establish the procedure as a relatively safe one when carefully carried out. I would point out here that there have been fatalities reported, too, a fact which should keep us reminded of the danger involved.

Agreeing, then, that there is a definite place for controlled hypotension, we have the responsibility of becoming familiar with it; in short, of learning when and how to use it.

It is not my intention to delve into specific details regarding the use of controlled hypotension, but rather to bring to your attention some of the general aspects of the subject and then refer you to others who are better able than I to inform you.

A brief review of certain aspects of the physiology of vascular pressure will remind us that the arterial blood pressure is dependent principally upon the cardiac output, the blood volume, the peripheral resistance, the blood viscosity, and the elasticity of the arterial walls. A change in any of these, too great to be controlled by natural compensatory measures, will affect the level of the blood pressure. For our present purpose, let us review the chain of events which occurs when hypotension is the result of reduction in the blood volume, and then the strikingly different pattern when hypotension is the result of a decrease in the peripheral resistance.

When blood volume is diminished, as in hemorrhage, a fall in the blood pressure is delayed for a time by compensatory peripheral vasoconstriction which tends to maintain the blood pressure at its previous level. Although this mechanism probably diminishes the adequacy of the capillary circulation, the temporarily sustained blood pressure is apt to conceal the failing state of circulation. At this point, intravascular fluids are introduced into the blood stream, another compensatory measure which partially replenishes the diminished blood volume and further obscures the precarious state of circulation. If hemorrhage continues to occur, however, compensatory measures fail, and there occur arteriolar and capillary dilatation with stasis and a fall in the venous pressure which impairs the venous return and diminishes the cardiac output. Finally, the capillary walls lose their integrity, and there is further reduction of blood volume by the passage of vascular fluids into the intravascular tissues. This is the chain of events which follows excessive reduction of blood volume and which may be referred to as "oligemic shock." All of us are familiar with this state and we have learned the gravity of the warning drop in the blood pressure and the serious consequences which follow if this chain of events is not promptly reversed.

A significantly different state exists, on the other hand, when the blood pressure is lowered by peripheral vasodilatation. In this state, the capillary circulation is much more effectively maintained, venous return should remain at a satisfactory level, and cardiac output is not seriously impaired. Provided there is adequate oxygenation and a sustained blood volume, the patient can withstand surprisingly low blood pressure, induced in this manner, without any apparent ill effect. We must point out, however, that if this vasoplegic hypotension is allowed to exist beyond the stage of fulfillment of basal nutritional needs of the tissues, then, once more, progressive failure in circulation will follow which will end in a fatality, if not reversed in time.

We must conclude, then, that the important difference in these two states of hypotension seems to be the adequacy or inadequacy of capillary circulation. In vasoplegic hypotension this adequacy can be maintained at a lower level of blood pressure than in oligemic hypotension.

In order to utilize the obvious advantages of lowered blood pressure during surgery with as little hazard to the circulation as possible, three major methods have been devised and used clinically.

First, there is the technic of blood withdrawal and replacement. Briefly, this procedure consists essentially of the withdrawal of blood from an artery until the desired level of hypotension is reached; the maintenance of this required level by periodic re-infusions and withdrawals; and the ultimate restoration to normal pressure by replacement. This technic, although used successfully, possesses certain inherent disadvantages. It requires moderately elaborate technical preparation. Also, execution must approach perfection, because even a minor technical failure which is not promptly corrected may result in disaster. In addition, we must remember that, as in simple blood loss, peripheral vasoconstriction will exist and, therefore, the blood pressure cannot be lowered with safety to the same extent as is possible when vasodilatation exists. With this technic, the blood pressure should not be allowed to fall below 80 mm. Hg., but it may be maintained at this level for several hours with a fair degree of safety. Because of its disadvantages, this method of controlling blood pressure is losing favor and is being replaced largely by safer, more easily executed methods.

Second, there is the technic of total spinal anesthesia. In this technic, the blood pressure is lowered as a result of peripheral vasodilatation which occurs as the sympathetic vasomotor fibres are blocked by spinal anesthesia. The vasomotor outflow leaves the spinal cord by way of all of the thoracic and the first two lumbar anterior roots. Therefore, the higher the anesthetic level is carried, the more extensive will be the sympathetic paralysis and the resulting fall in blood pressure. Blocking above the first thoracic segment affords no further advantage and is undesirable because respiratory paralysis may occur. Preparation must be made to assist or to maintain ventilation artifically in case such a situation does develop. In addition, when vasomotor control is paralyzed, there exists a state of postural hypotension. The reflexes which ordinarily initiate compensatory vasomotor control on changes in posture are abolished. Then, on elevation of the head, the arterial pressure falls markedly and the venous blood is drained away from the head by gravitation. In the dependent regions the blood tends to become pooled. Because of these profound changes, care must be exercised neither to elevate nor to lower the head excessively, lest the cerebral circulation become inadequate. It is, of course, imperative that the blood volume be sustained and a high oxygen concentration maintained. Using this method, a reduction of blood pressure to 50-60 mm. Hg. may be tolerated for several hours, since the co-existing vasodilatation helps to sustain the capillary circulation. Bloodless operative fields can be attained, but this procedure also requires careful preparation and execution and is not entirely controllable or safe. Therefore, it, too, is largely being discarded.

Third, there is the more favored technic of lowering the blood pressure by the intravenous injection of sympathetic ganglionic blocking agents. This technic has become very attractive because of the simplicity of administration and the relative ease of control. My associates and I have used Bistrium (hexamethonium bromide) for this purpose in selected cases for several years and have had no evident complications. In general, blood loss has been considerably reduced and operative procedures significantly facilitated and shortened. There are, however, some problems associated with its use.

Careful attention must be given to the dosage, as it varies widely, and the effect is, to some extent, unpredictable. The young patients, and those requiring excessive drugs for anesthesia, are more apt to require larger doses. From 12.5 to 25 mgs. may be tried initially and additional amounts given fractionally depending upon the response. There appears to be a tendency to develop a tolerance, so an effective initial dose is desirable. Once sympathetic ganglionic block has been achieved, any additional dosage will not lower the blood pressure further.

Since postural hypotension, which is entirely similar to that following total spinal anesthesia, is produced with the use of Bistrium, additional control is possible by varying the elevation of the head. That is, elevation of the head will further reduce the blood pressure, whereas lowering the head will, to some extent, raise the pressure. In this connection, however, one must keep in mind the great reduction in cerebral blood flow which will accompany the elevation of the head, and such elevation should not exceed 15 to 30 degrees if the blood pressure is excessively low. There is no accurate way of being sure of adequate cerebral circulation, though it can be fairly well estimated by the color of the face and scalp and by the capillary refilling time. When the brain is exposed, an experienced surgeon can judge the adequacy of the circulation by its appearance.

There seems to be little advantage in dropping the blood pressure below 60 mm. Hg, and 80 mm. is sufficient for most procedures. This range of pressure can be maintained for several hours with a fair degree of safety, provided all other factors are controlled. Should the blood pressure drop too low, it can usually be restored by lowering the head, or, if this fails, by infusing a vasoconstrictor drug such as Levophed ® (Norepinephrine).

Another phase of this technic which must be emphasized is the maintenance of adequate blood volume, for if this is allowed to diminish, serious shock may ensue. Any blood loss must be kept minimal and rapidly replaced. Bistrium must never be given if the blood pressure has already fallen as a result of blood loss.

Oxygenation is extremely important and must be kept maximal at all times.

Pulmonary ventilation must be kept adequate, also, and either assisted or controlled as indicated. Cyanosis must be avoided and the capillary refilling time must not be delayed too long.

After the need for hypotension has ended, it is necessary to restore the blood pressure to a normal level before the wound is closed, and any bleeding which results from the rise in pressure must be meticulously controlled to avoid post-operative hemorrhage. It is important, also, to continue oxygenation and blood pressure control throughout the entire recovery period.

As we consider the use of these various hypotensive technics, let us be ever mindful of the need for careful judgment in their employment. That danger is inherent in the very nature of the disturbed physiology is attested to by the recorded experiences of the pioneers in this field. The routine use of hypotensive technics for the casual convenience of the surgeon must be condemned. Selection of cases for hypotension should be the result of sound judgment, with preference given always to the overall plan offering the least calculated risk to the patient. Arteriosclerotic, hypertensive, and other poor risk patients must be handled very cautiously and in these cases especially, experienced judgment must be exercised. In general, they are not the patients who would be selected for marked lowering of the blood pressure because of the narrower margin of safety.

Let us say, then, that despite the hazards which are involved, controlled hypotension is basically sound and will remain as an aid to surgery, though the technic will undoubtedly be improved.

It would be unwise to leave this subject without mentioning some other relatively simple, but very practical, means of controlling vascular pressures.

One of the more obvious, and yet more effective, methods is that of simply elevating the operative region above the level of the heart. I am sure that many of you routinely avail yourselves of this postural advantage.

Arterial pressure is reduced by this method in varying degrees, dependent upon the condition of the subject. We know that its fall is minimal in an unanesthetized patient due to the compensatory reflexes functioning to maintain a near-constant blood flow. On the other hand, in general anesthesia, these reflexes are less efficient so that, to some degree, there exists a postural hypotension. Therefore, it is well to remember that, with postural elevation, arterial pressure falls slightly in the normal unanesthetized subject, moderately during general anesthesia, and there will be a profound drop if the sympathetic control has been blocked.

Venous pressures present a somewhat different picture. Since veins are essentially free of neural control, venous pressure is considerably influenced by gravity, and we find that elevation causes a profound drop regardless of other factors. From a practical standpoint, elevation is a *most* effective means of controlling bleeding of venous origin. In pioneer days, neurosurgeons had great fear of entering a venous sinus within the cranium, for hemorrhage was sure to be profuse, if not entirely uncontrollable. Matters were worse if the lesion had caused an increase in intracranial pressure. Surgeons now realize that simply by elevating the head, venous hemorrhage is no longer a serious problem. It should be noted here that because of this striking advantage of elevation of the head, the sitting position gained some degree of popularity in operations about the head and neck. Many surgeons, however, after an initial period of enthusiasm, have discarded this position because of fatalities resulting from serious falls in blood pressure and from air embolism. Normally, the venous pressure in the head drops to plus or minus zero in the erect position and embolism may occur if there is venous inspiration of air at a time when the venous pressure has attained a negative value. Therefore, it has been realized that, if the sitting position is avoided and the patient is arranged so that the degree of elevation can be controlled by tilting the table, these hazards will not be present. Air embolism can probably be avoided if the head is not elevated more than 65 degrees from the horizontal position.

Venous pressures are greatly influenced by other factors as well. Sudden changes in intrapulmonary pressures caused by coughing, straining, or vomiting will increase bleeding markedly during the positive phases of venous pressure and, of course, invite air embolism if a negative fluctuation occurs. It, therefore, becomes desirable to maintain an unobstructed airway, gentle respiratory excursions, and a minimal intrapulmonary pressure.

It is obvious that venous pressure will be increased if there is any obstruction of the venous return. This should be avoided, therefore, by removing any pressure on the large veins which drain the extremities and the head, and by eliminating any compression of the abdomen. Excessive flexion or extension of the neck will cause venous compression and should be avoided also.

Venous pressures are further influenced by the state of skeletal muscle tone so that muscle relaxants, such as Anectine, (R) lower vascular pressures, and consequently, diminish venous bleeding. The use of Anectine (R) has the further advantage of lessening or precluding coughing, straining, or vomiting at a time when such an occurrence might be disastrous, such as during the dissection and exposure of an intracranial aneurysm.

Another practical aspect of vascular pressure which should not be overlooked is the large quantity of blood which is contained within the vascular reservoir of the extremities. The effective blood volume may be greatly increased or diminished quickly by such maneuvers as elevating or lowering the extremities or by compression bandaging. This is particularly true when sympathetic paralysis exists. The elimination or the use of abdominal compression may be similarly employed to lower or raise the general blood pressure.

Finally, the reduction of cerebrospinal fluid pressure, by spinal or ventricular drainage, may strikingly reduce bleeding in cranial and spinal procedures and may be utilized at the discretion of the operating surgeon.

Thus, we see that, in addition to the more complicated methods of controlling intravascular pressures, there are many fairly simple measures which may be used to great advantage in controlling hemorrhage and which require only a little ingenuity and a practical understanding of vascular physiology.

The time allotted has not permitted covering many of the details of this complex subject. It is my hope that you have found this rather general discussion provocative, and that your interest has been so stimulated that you will become more familiar with the physiology of vascular pressure and the controls which may be applied, for

(Continued on page 67)

Muscle Relaxants

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The use of muscle relaxants in anesthesia is one of the most important advances that have been made in this specialty in at least ten years. It was only in 1942 that Griffith and Johnson reported on the use of "Intocostrin" in 25 cases[1] although it had been used as a laboratory drug for many years and clinically in the treatment of convulsive states. Much has been learned about muscle relaxants and much progress has been made in their use since then.

Intocostrin was a purified extract of curare obtained from chondodendron tomentosum. The activity of Intocostrin was known to be due to a crystallizable substance, d-tubocurarine chloride. It was only natural that before long a solution of d-tubocurarine chloride was introduced for use in anesthesia and replaced the use of Intocostrin. It was stated early that the principal mechanism of action was the blocking of the response to the nicotinic action of acetylcholine (including depression of conduction through all autonomic ganglia) and that physostigmine and prostigmine were antagonistic to the action of curare.[2]

Pharmacological studies of the action of curare were continued. To understand the action of curare and the other relaxants, it is necessary to know something of the physiologico-pharmacological changes that take place when a muscle contracts.[3] The neuromuscular unit is the site that is of importance to anesthesiologists. In the resting state, the post-junctional membrane, which is actually between the nerve axon and the muscle fiber and frequently called the motor end plate, is said to be polarized. That is, there is a row of positive charges on one side of the membrane and a row of negative charges on the other side. When the nerve is stimulated, secretion of acetylcholine takes place in the subneural space lying between the nerve and the post-junctional membrane. Acetylcholine depolarizes the membrane; i.e., positive and negative charges are equally distributed on each side, and muscular contraction is hereby initiated. Restitution of the neuromuscular unit takes place by acetylcholinesterase

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destroying the acetylcholine, and the membrane becoming repolarized. The neuromuscular unit is then ready for transmission of the next impulse.

When d-tubocurarine chloride is administered, the depolarizing action of acetylcholine is blocked so muscular contraction does not take place. Physostigmine and prostigmine (neostigmine) antagonize curare by inactivating acetylcholinesterase and allowing the acetylcholine to accumulate to a concentration which will cause depolarization, and thus, muscle response. Physostigmine and prostigmine also are mild competitors with curare for the cholinergic receptors at the end plate; i.e., the post-junctional membrane. Tensilon, another curare antagonist, produces a major portion of its anticurare effect by competition with curare for the cholinergic receptors, and produces a subsequent displacement of curare. Tensilon has a direct stimulating effect on the muscle through its action on the end plate, but an overdose causes a persistence of polarity, prohibiting the normal change from polarization to depolarization, and thus acts as a muscle relaxant itself. Although no minimum dosage has been established, from clinical experience it seems wise not to administer more than 50 mg. to a patient.

Although it was stated early that curare caused a blockade of transmission through all autonomic ganglia, it is believed now that in the doses used clinically, no significant blockade of ganglionic transmission is effected.

When curare is administered intravenously, a rapid fall in plasma concentration occurs soon after injection.[4] After 15-20 minutes, the plasma concentration decreases very slowly and the d-tubocurarine chloride remains in the plasma for several hours. There is evidence that the preponderance of curare disappears from the blood stream into muscle, and the liver and kidneys receive appreciable amounts. It is partially destroyed in the liver, but largely eliminated, unchanged, in the kidneys. However, liver or kidney disease does not seem to intensify or prolong the action of curare. No direct effect on the heart has been noted, although large amounts given intravenously in a short period of time occasionally produce a momentary fall in blood pressure.

Curare is selective in action, affecting the muscles of the head and neck before progressing to the muscles of the extremities and abdomen. The muscles of respiration, and finally the diaphragm, are the last to be affected. Although it sometimes seems to have analgesic action, actually curare has none.

The muscle relaxants, as used in anesthesia, are always administered intravenously. The effect of curare is obtained within 1-2 minutes, although usually about 5 minutes are required for its maximum effect. The action persists for 20-30 minutes and there is little or no cumulative effect if the drug is administered again within an hour or two. These times must be considered in determining when the curare is to be administered, varying with the speed of the surgeon in getting the abdomen open, with the duration of the procedure, and with the time required for closing the abdomen. The dose of curare will vary with the individual, but it usually requires 80-120 units (12-18 mg.) to relax an abdomen when cyclopropane or pentothal-nitrous oxide is the inhalation anesthetic being used. It is advisable to start with 6-9 mg. and administer additional 3 mg. doses until the desired relaxation is obtained. If ether is the primary agent about one-half of this amount is required. Depending upon the length of the procedure, additional doses may be necessary during the course of the operation and to close the abdomen. For closure, usually only one-half to three-fourths of the original relaxing dose is required.

When optimum relaxation of the abdomen is obtained, some depression of respiration is usually present also. Complete intercostal paralysis and partial diaphragmatic paralysis produces a shallow, jerky respiration. When this occurs, complemental respiration should be administered and continued until adequate respiration returns, which is usually within 5-15 minutes. Complemental assistance to respiration is better than completely controlled respiration, for this allows some negative pressure to be produced in the thoracic cage and thus aids in the venous return to the heart. If apnea is produced, controlled respiration will be necessary. If respiratory activity is not adequate at the termination of the operation, the safest procedure is to aid respiration until sufficient curare has been removed from the blood stream and no depression of respiration remains. One of the anticurare drugs, such as Tensilon or neostigmine, may be administered. These drugs should not be administered to a patient who is apneic, but only after respiratory movements have begun to return spontaneously. One must realize, also, that the effect of the latter drugs may wear off before the effect of the curare, and the patient then returns to a state of deficient respiratory activity. Cullen[4] suggests that no patient be dismissed from the attention of the physician, after the administration of Tensilon to relieve curare paralysis, until he can voluntarily raise his head and hold it elevated for at least 10 seconds. He states that once patients have reached this point of decurarization, there seems to be little regression. Some deaths following the use of curare have been reported, atelectasis being the cause of death.[5] These have been attributed largely to returning the patient to his bed before the intercostals have recovered.

A toxic effect of curare which may also play a part in the development of atelectasis is bronchoconstriction. The bronchospasm produced by curare is similar to that produced by histamine, and it has been shown that curare does liberate histamine from body tissues.

The transplacental transmission of curare was questioned, since this drug was advocated for use in Cesarean sections. Originally it was reported that curare did not cross the placental barrier, but more recently it has been determined that transmission does take place. However, in the doses used clinically, the concentration in the fetal blood streams is not sufficient to be detected by the methods usually employed or to cause any respiratory depression.[4]

There are only two contraindications to the use of curare in anesthesia. One is myasthenia gravis. This condition is aggravated by the injection of curare. The other is the inability to perform artificial respiration. One can never be sure when respiratory depression is going to occur with the use of curare, so some method of administering artificial respiration, including intratracheal intubation if necessary, must be available at a moment's notice.

With the widespread interest in the use of curare for muscle relaxation, it was only natural that investigations were undertaken to find other compounds which also had the power of relaxing muscle. As the investigations proceeded, it was discovered that, with few exceptions, the compounds which manifested this power had one chemical characteristic in common: they all were drugs which contained more than one quarternary ammonium group 15 angstrom units apart in the molecule.[3]

Gallamine triethiodide (Flaxedil) was one compound which gained fairly wide usage. The action of gallamine is similar to curare in preventing the depolarizing action of acetylcholine. Like curare it has no effect on the central nervous system, and, in clinically used doses, does not affect the transmission through autonomic ganglia. It is also eliminated by the kidneys. Gallamine does not diminish blood pressure, vascular tone, or myocardial activity, but it does possess an atropine-like effect on the cardiac vagus, although not on the vagus innervating the intestine. Consequently, an increase in cardiac rate does follow its administration. Gallamine was reported to have no discernible histamine-like action (bronchospasm) but it was found later to liberate one-fifth to one-half less histamine than d-tubocurarine.[6] The effect on respiration is similar to d-tubocurarine, but not as severe, in that relaxation can be obtained with less respiratory depression.

Myanesin is a different type of compound — a complicated derivative of propane — which was introduced by the British as being a synthetic curarizing agent superior to curare because it had a wider margin of safety. Abdominal relaxation was obtainable even in the conscious patient, and the drug did not cause intercostal paralysis in producing full relaxation of the abdomen. However, investigators in Canada[7] and the United States concluded that this preparation had no advantages over curare. In fact, there were some disadvantages: the relaxing effect was less predictable, occasionally local irritation was set up in the vein at or above the site of injection, hemoglobinuria associated with albuminuria occurred, and severe cardiac depression or heart block in animals was produced. Consequently, its use was limited.

Benzoquinonium - mytolon, is another compound with the usual quarternary nitrogen atoms about 15 angstrom units apart. However, its mode of action is not exactly clear. It has properties similar to d-tubocurarine, but also exerts effects similar to decamethonium, which will be discussed in the following section. Probably the major portion of its effects are similar to d-tubocurarine. It is especially interesting from a pharmacologic standpoint because of this dual mechanism of action. Its most notable characteristic is the red color of the crystals and the solution. Its first clinical use was reported by Arrowood.[9] The potency of mytolon equals or exceeds that of d-tubocurarine, while with equipotent doses, the toxicity equals, or is less than, that of the latter drug. Curarizing doses do not cause any significant change in pulse rate or blood pressure and are without direct cardiac toxicity. No effect on autonomic ganglia or the vagus has been demonstrated. Adequate muscular relaxation is accompanied by definite respiratory depression, which is characterized by a decrease in amplitude, without the jerky, spasmodic type of respiratory effort that accompanies d-tubocurarine. There is no prolonged respiratory depression. No evidence of bronchoconstriction has been noted. Its chief advantages are its lack of effect upon the cardiovascular system and the prompt recession of respiratory depression.

Decamethonium (C-10, syncurine) produces its effect in a manner different from the previously discussed relaxants. It produces persistent depolarization of the postjunctional membrane, and thereby prevents the physiological depolarization - repolarization sequence caused by the acetylcholine-cholinesterase system that is necessary for unimpaired neuromuscular transmission. The anticurarizing agents (tensilon and neostigmine) are thus ineffective against decamethonium, and no effective counter agent has been introduced, although d-tubocurarine and its related compounds are antagonistic to this type of relaxant.

Decamethonium acts much faster than d-tubocurarine, reaching a maximum in 2-4 minutes, beginning to wear off in 8-10 minutes, and completely disappearing in 15-20 minutes.[10] Theoretically, there should be no difficulty with postanesthetic respiratory depression using such a short-acting drug; but actually there have been numerous incidences of respiratory depression lasting an hour or more following anesthesia. To the writer's knowledge, the cause of this is hereto unexplained. The only treatment for such a condition is to administer artificial respiration until adequate spontaneous respiratory activity returns.

Respiratory depression accompanies adequate abdominal relaxation, as with the other relaxants. One advantage of decamethonium is the absence of liberation of histamine from the body tissues, and thus absence of bronchospasm, hypotension or capillary damage. It produces no deleterious effects on circulation.

Decamethonium is administered in 1 to 2 mg. doses until the desired relaxation is obtained. For abdominal relaxation, about 5 mg. are usually required, and, due to its relatively short action, subsequent doses are necessary. Some writers believe that abdominal relaxation can be obtained without respiratory depression, but most believe this is not true, although it is not necessary to carry the patient to complete apnea.[11] It does possess the disadvantage that occasionally its muscle-relaxing effect wears off abruptly. This, at times, can be troublesome to the surgeon.

Decamethonium was used extensively for tracheal intubation. It is miscible with pentothal, its action is rapid and not too prolonged and, according to some,[12] sustains the vocal cords in abduction. However, since the introduction of succinylcholine, its use has greatly diminished. It is still a useful agent to be administered in a single dose for the production of muscular relaxation of relatively short duration; for example, for endoscopy or for peritoneal closure in cases in which the effect of spinal anesthesia has worn off.

The latest, and in the writer's opinion, to date the best muscle relaxant is succinylcholine. As Little, Hampton and Grosskreutz state:[13] "The ideal muscle relaxant should be capable of very brief action, for use in such manipulations as intratracheal intubation, electric shock therapy and the reduction of fractures; it should be capable of action of sufficient duration to permit prolonged muscular relaxation during major surgical interventions; it should interfere in no way with normal metabolic processes or with the normal patterns of respiratory or cardiovascular action; its relaxing action should be followed by the prompt resumption of muscular reactivity at the termination of operation; it should be rapidly metabolized in the body to innocuous degradation products, and this despite the presence of any pre-existing disease or pathologic condition in any organ system; and it should accomplish all of this without the manifestation of either toxic side-effects or complications." The fulfillment of all these criteria by any one drug would appear doubtful, but succinylcholine comes close to that ideal on a number of different counts, mainly because a total relaxant dose is fully dissipated within 2-3 minutes.

Succinylcholine, which is 2 molecules of acetylcholine linked together, affects muscle physiology like decamenthonium; i.e., produces a persistent depolarization at the post-junctional membrane. Due to this depolarization, muscular fasciculations occur, especially when the drug is administered rapidly. As with decamethonium, an antagonism exists between succinylcholine and drugs acting at the myoneural junction as do d-tubocurarine and gallamine. The administration of succinylcholine during recovery from curarization will diminish the relaxation. Also, neostigmine and other anticholinesterase drugs cannot be employed as antidotes to succinylcholine, as they will actually prolong the neuromuscular block produced by that drug.

The short duration of action is due to the hydrolyzation of succinylcholine by the pseudocholinesterase of the plasma, and more slowly by the true acetylcholinesterase of the red cells into succinylmonocholine and choline. Then, succinylmonocholine is hydrolyzed to succinic acid and choline.[14] Succinylmonocholine is a weak depolarizing-type inhibitor of neuromuscular transmission. Only in those individuals having a low plasma titer of pseudocholinesterase is the action of succinylcholine prolonged. Procaine also has some prolonging effect on succinylcholine as it, too, is hydrolyzed by pseudocholinesterase and thus competes with succinylcholine for this enzyme.

Clinically, the muscles are affected in the same order and manner by succinylcholine as by curare preparations; i.e., muscles innervated by the cranial nerves are affected first, followed by the peripheral muscles, the intercostal muscles and finally the diaphragm, although the effect is so rapid that this sequence is rarely noted.

The maximum effect of succinylcholine is reached in 30-60 seconds and recovery is usually complete in 2-6 minutes. It is this rapid effect and recovery which gives excellent control of both degree and duration of relaxation on a minute to minute basis. Another advantage is its relative sparing action on respiration. With comparable muscular relaxation there is definitely less diminution of respiratory depth; good relaxation, together with adequate respiratory exchange, frequently being obtained without the use of assisted respiration. The respiratory movements are smooth in contrast to the jerky respirations frequently noted with the other muscle relaxants. No prolonged post anesthetic depression has been encountered with succinylcholine alone. There seems to be a tendency for a slight elevation of pulse rate, due to a possible mild vagolytic effect, and blood pressure may rise slightly. Salivation, excessive bronchial secretions, laryngeal spasm, bronchospasm and other evidence of histamine release have not been observed. There is no indication of blocking or stimulating action on the autonomic nervous system.[15] The low toxicity of succinylcholine has been demonstrated in animals.[13] Dogs, maintained on artificial respiration, tolerated doses which were 450 times the paralyzing dose, whereas 15 times the paralyzing dose of d-tubocurarine chloride proved to be lethal.

For abdominal surgery, the continuous intravenous drip method is by far the most practical and efficient. The succinylcholine can be made up in any concentration, but a 0.05 to 0.1 per cent concentration (0.5 to 1 mg. per cc.) in glucose or saline solutions is most common. This is introduced into the patient's vein and then he is put to sleep with pentothal or inhalation anesthesia. If intubation is to be performed, the drug may be dripped rapidly until relaxation sufficient for intubation is obtained. If not, it should be dripped slowly until about 30-60 seconds before the peritoneum is opened and then rapidly administered. Apnea may ensue before one is sure of having obtained sufficient abdominal relaxation, but this may be counteracted by controlled respiration and the drip slowed until the optimum rate is found—one which will give sufficient relaxation and respiratory activity. In some cases it may be necessary to complement respirations. If so, it is physiologically prudent to assist every other inspiration so that the negative pressure of inspiration, with its support of venous return to the heart, will not be interfered with completely. While exploration of the abdomen is being performed, and until the intestines are packed off or retractors put into place, it is necessary to keep the patient well relaxed. No specific dose can be recommended to accomplish this, for the administration of succinylcholine must be individualized even more than the other relaxants. The dose may vary from 0.6 to 7.5 mg. per minute depending upon body weight, muscle tone, and plasma cholinesterase activity. The average dose has been found to be 2.5 mg. per minute. When the aforementioned procedures are completed, it is usually possible to diminish the rate of administration to about one-third of the initial rate until it is time to close the peritoneum. At this time it will be necessary to increase the dose of succinylcholine in order to obtain sufficient relaxation for unhampered closure of the peritoneum. Relaxation should be comparable to spinal. As soon as the peritoneum is closed, the rate of administration can again be diminished, or, in some cases, stopped entirely. It is wise to leave the succinylcholine dripping very slowly until the fascia is almost closed, for occasionally the relaxing effect wears off so fast that the surgeon is not able to approximate the fascia readily, and a small additional amount of succinylcholine will be necessary. The total amount required for an operation varies with the individual patient and the duration of surgery. It may be as little as 100 mg. and it has been over 1000 mg. If most of the prepared succinylcholine in glucose or saline is not used, the flask may be covered with an alcohol sponge and the mixture stored in a refrigerator. However, it is believed that the potency of the diluted succinylcholine decreases during storage. Due to this fact and to the danger of contamination, it is advisable to use the mixture within 48 hours.

Succinylcholine has replaced decamethonium for intubation with pentothal, due to the rapid and profound effect upon both the muscles of the jaw and the glottic stuctures, and due to the recovery of spontaneous respiration within seconds or minutes. For this procedure, the writer believes it is still wise to topically anesthetize the larynx, either by spraying or by injecting a topical agent. Some men do not think this is necessary.[16] Oxygen should be administered in order to build up the oxygen reserve of the tissues and thus provide a longer safe period for the intubation. This may be done with the patient awake or after injecting a small amount of pentothal so that the patient will not be aware of a mask being placed on his face. After 4-5 minutes of breathing 100 percent oxygen, pentothal and succinylcholine are injected. If these are to be mixed in the same syringe, it is necessary to do so just before they are administered, for the basic pentothal inactivates the succinylcholine. Usually from 10-30 mg. of succinylcholine are necessary for this procedure. This dosage will produce first the fasciculations referred to previously, then muscular relaxation, and usually apnea. (The termination of the fasciculations is an indication of the optimum time for intubation.) While waiting for relaxation, the administration of oxygen should be continued and one may even hyperventilate the patient. It may be desirable to obtain apnea which provides ideal conditions for intubation, obviating the necessity for too much haste, and subsequent trauma, and preventing bucking at the time of the introduction of the intratracheal catheter. After intubation, the patient is ventilated with 100 per cent oxygen first, and then with the anesthetic mixture one intends to use for maintenance. Respirations are controlled or assisted until resumption of full, spontaneous respiratory activity.

Single intravenous injections may be used in other instances. When d-tubocurarine has been used and the effect has been dissipated, succinylcholine may be used for the momentary relaxation necessary for peritoneal and abdominal wound closure. A 10-20 mg. dose will produce sufficient relaxation of the rectal sphincter to prevent the recto-laryngeal reflex which frequently takes place. Relaxation of muscles to allow reduction of a fracture may be obtained with succinylcholine, but the injection should be made at a moderate rate in order not to produce too great muscular fasciculations. Laryngospasm which does not terminate following the removal of the causative stimulus, if known, will usually yield to the administration of 10 mg. of succinylcholine.

The advantages of succinylcholine — its easy controllability; the absence of apnea during, and of prolonged respiratory depression following its administration; and the paucity of the side effects that accompany its use — make it the muscle relaxant of choice for most cases.

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Anesthesia in Geriatric Surgery

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Today there are more people living beyond the age of 65 years than ever before. It is of interest to note that the mean length of life in early Rome was between 20 and 30 years. Just one hundred years ago the mean length of life was 40 years. It has been predicted by Dublin that the mean length of life in 1975 will be 71.5 years.[1]

It is the opinion of many surgeons that few operations are too hazardous to be considered for the elderly patient, provided he is properly prepared and managed during the operative and post-operative periods. However, Cole[2] has pointed out that in this age group the operative mortality is almost three times as high for emergency surgical procedures as it is for elective operations.

This article will present an analysis of the anesthetics administered to patients 70 years of age or over at Butterworth Hospital in Grand Rapids, Michigan during the period from January 1, 1949 through December 31, 1953. Anesthetics during this five year period were administered by nurse anesthetists, resident physicians, and the director of anesthesia.

In preparing this article 748 anesthesia records were analyzed. There were 22 anesthetics given to patients of 90 to 97 years. 161 anesthetics given to patients of 80 to 89 years, and 565 anesthetics given to patients 70 to 79 years.

SEX

In the total series there were 421 male patients and 327 female patients.

The distribution as to sex in this series is found in Table 1.

PHYSICAL STATUS

Evaluation as to the physical status of the patient was recorded using the code as described by Collins.[3] A patient with physical

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status of 1 is a patient having no physical disability besides that requiring operation. Physical status 2 is a patient with one physical disability besides that requiring operation, i.e. hypertension. Physical status 3 is a patient with two physical disabilities besides that requiring operation, i.e. emphysema and diabetes. Physical status 4 is a patient with three physical disabilities besides that requiring operation i.e. hypertensive heart disease, asthma, anemia. Number 5 is an emergency patient good risk, 6 is an emergency patient poor risk, and 7 is a moribund patient whose only chance of survival is an operative procedure.

As is noted in Table 2, the majority of the cases were at least a risk 2. The next largest group were risk 3 and a surprising few were risk 4.

On further analysis of the physical disabilities encountered, the usual expected degenerative processes were present. Those occurring most frequently are listed in Table 3.

Pre-operative complications encountered and listed by the various systems of the body are in Table 4.

Anesthetic Agents and Methods

The anesthetic agents and methods used are listed in Table 5. With the advent of combined anesthesia and curarizing agents and the acquired skill in the use of these methods and agents there was an increased number of patients receiving combined agents from 1951 through 1953.

Premedication

Premedication most frequently used in this series was demerol (meperidine) and atropine or scopolamine. Atropine was given by choice in patients that were senile (Cerebral arteriosclerosis) and male patients having urological surgical procedures. Short acting barbiturates i.e. pentobarbital (nembutal) were used sparingly to avoid mental confusion postoperatively.

In most instances the premedication given was satisfactory. Only a small number of patients were insufficiently premedicated.

Pentothal Dose

The patients in this series tolerated pentothal well. In all cases the initial dose of pentothal (using 2-½% concentration) was reduced to avoid profound initial respiratory depression. The majority of the patients took up to one gram of pentothal.

Curare Dose

D-Tubocurarine or flaxedil (Gallamine) were the curarizing agents used. These drugs were used to accomplish relaxation for operative procedures, to achieve intratracheal intubation, and to increase bladder capacity. Table 8 indicates the total doses used.

Anesthesia Time

The total time of anesthesia for two-thirds of the cases was under 90 minutes. The longest case of the 80 year olds was 3 hours and 30 minutes and the longest case for the 70 year olds was 4 hours and 45 minutes. The total anesthesia time is noted in Table 9.

ANESTHESIA COMPLICATIONS

Table 10 shows the most common anesthesia complications encountered.

In view of the high incidence of emphysema and chronic cough it is not surprising that 35 of the patients coughed during anesthesia.

Bronchospasm occurred in four cases—two from vomiting with aspiration, one under pentothal and one under pentothal-curare. The latter two cases responded promptly to ether anesthesia.

There were twelve incidences of laryngospasm. All were corrected with oxygen under positive pressure and changing of agents. It is interesting to note that although the majority occurred under pentothal there were three that developed under ether anesthesia.

Under arrhythmias, change in pulse rate such as bradycardia and tachycardia, as well as change in pulse rhythm was included.

The majority of cases of hypertension in this series appeared to be associated with the use of curare. In each instance a marked hypertension was noted shortly after the initial dose of curare was given and immediately returned to normal range when an antagonistic drug was administered. However, one cannot overlook the possibility of carbon dioxide accumulation as assisted respirations were the exception rather than the rule.

Hypotension in this series was associated with reflex from the operative field, deep anesthesia and spinal anesthesia. In each instance vaso-pressors were used sparingly and in small doses.

There were only two deaths in the operating room in this entire series. One patient had a massive mesenteric thrombosis, was in shock, and received only oxygen therapy. During surgery the surgeon encountered uncontrollable bleeding and the patient expired. The other patient, a 75 year old negro house wife with a large right kidney mass and history of occasional pre-cardial pain, withstood a very traumatic and difficult removal of a large carcinomatous kidney throughout two hours. The patient had been in shock due to blood loss, which had been replaced and the circulation had stabilized at a normal level. As the skin sutures were being inserted her heart suddenly ceased to beat. It was believed she died of a coronary occlusion. No autopsy was obtained.

The largest number of cases were done for correction of urological pathology.

Most of the abdominal surgery was done for gall bladder disease or carcinoma of the abdomen.

It was interesting to note that out of the 97 hip nailings 51 were in the 80 and 90 year old age groups.

The most common post-operative complications were urinary retention and intestinal distention.

Due to the stirr-up regime and minimal use of analgesics the incidence of bronchopneumonia of those who survived was only 3.

TABLE 1

Sex Distribution

	90 years	80 years	70 years	Total
Males	6	95	320	421
Females	16	66	245	327

TABLE 2

Physical Status Preoperative

Code Number	90 years	80 years	70 years	Total
1	0	6	48	54
2	5	62	281	348
3	13	62	172	247
4	0	14	18	32
5	0	4	6	10
6	4	13	35	52
7	0	0	5	5

TABLE 3

Common Degenerative Processes Preoperative

	90 years	80 years	70 years	Total
Emphysema	11	59	203	273
Hypertrophy prostate	5	21	120	146
Hypertension	2	40	152	194
Arteriosclerotic heart disease	14	45	172	231
Hypertensive heart disease	4	24	77	105
Cerebral arteriosclerosis	13	35	89	137
Diabetes mellitus	2	7	26	35

TABLE 4

Preoperative Complications

	90 years	80 years	70 years	Total
Respiratory	16	81	331	428
Circulatory	20	132	423	572
Genitourinary	5	35	173	213
Gastro-intestinal	2	23	101	126
Neurological	13	37	100	150
Metabolic	8	40	103	151

TABLE 5

Anesthetic Agents and Methods Used

	90 years	80 years	70 years	Total
Nitrous oxide-oxygen	1	6	8	15
Nitrous oxide-oxygen-ether	12	49	91	152
Nitrous oxide-oxygen-ether-curare	0	1	10	11
Pentothal-nitrous oxide-oxygen	2	31	137	170
Pentothal-nitrous oxide-oxygen-ether	1	5	35	41
Pentothal-curare-nitrous oxide-oxygen	1	18	119	138
Pentothal-curare-nitrous oxide-oxygen-ether	1	5	42	48
Nembutal-nitrous oxide-oxygen	0	0	8	8
Nembutal-nitrous oxide-oxygen-ether	2	8	20	30
Spinal	1	31	75	107
Local	1	7	20	28

Note: Curare, or curare-like drug, used was D-tubocurarine or flaxedil.

TABLE 6

Premedication

	90 years	80 years	70 years	Total
Demerol-atropine or scopolamine	18	121	403	542
Morphine-atropine or scopolamine	2	13	81	96
Demerol-atropine or scopolamine-barbiturate	0	19	68	87
Miscellaneous	2	7	10	19
None	0	1	3	4

TABLE 7

Total Dose of Pentothal Used

	90 years	80 years	70 years	Total
To 1 gram	6	9	265	280
1 to 2 grams	0	3	57	60

TABLE 8

Total Dose of Curarizing Agents Used

	90 years	80 years	70 years	Total
*to 40 units	2	14	69	85
40 units to 100 units	0	9	81	90
100 units plus	0	0	22	22

*D-tubocurarine 20 units = 3 mgm.

TABLE 9
Time of Anesthesia

	90 years	80 years	70 years	Total
To 30 minutes	4	30	121	155
30 min. to 60 min.	9	51	171	231
60 min. to 90 min.	6	46	122	174
90 min. to 120 min.	3	19	84	106
3 hours plus	0	15	67	72

TABLE 10
Anesthesia Complications

Coughing	35	Arrythmia	41
Bronchospasm	4	Hypertension	42
Laryngospasm		Hypotension	67
Pentothal	9	Vomiting	16
Ether	3	Deaths in O.R.	2

TABLE 11
Types of Surgery

	90 years	80 years	70 years	Total
Upper abdominal	1	11	80	92
Lower abdominal	3	33	74	110
Perineal G.U. and Gyn.	3	54	258	315
Miscellaneous majors Kidney & Abd. perineal	0	0	10	10
Extremity Fractured hip, leg amp. etc.	12	44	65	121
Operations about head	2	7	38	47
Thoracic	0	3	17	20
Minor surface	1	9	23	33

TABLE 12

Patients Receiving More Than One Anesthetic

No. of Anesthetics	90 years	80 years	70 years	Total
2	3	13	57	73
3	0	3	9	12
4	0	0	4	4
5	0	0	2	2

TABLE 13

Anesthetics Used In Patients Who Died In First Eight Days

	90 years	80 years	70 years	Total
Nitrous oxide-oxygen-ether	0	3	1	4
Pentothal-curare Nitrous oxide-oxygen	0	0	4	4

DEATHS

Finally, of the entire series there were 13 deaths or 1.76% mortality within the first eight days. There were no deaths in the 90 year olds. There were 4 deaths, or 2.48% mortality of the 80 year olds. There were 9 deaths or 1.59% mortality of the 70 year olds.

Of the patients who died in the first eight days, half received nitrous oxide, oxygen and ether. The other half received combined anesthesia. (note Table 13.) Of the 67 patients requiring emergency surgery, there were five who died. Two received nitrous oxide, oxygen, ether, one received pentothal-curare and nitrous oxide-oxygen, one received only oxygen therapy, and one was done under local anesthesia.

SUMMARY

A series of 748 cases was analyzed over a five year period in the age group of 70 years and upwards. As might be expected the analysis of the physical status of this age group revealed the usual degenerative diseases. Hypertension and heart disease were most commonly observed. Emphysema frequently associated with asthma, chronic bronchitis and bronchiectasis were the usual respiratory complications.

Premedication was usually demerol and atropine or scopolamine. Barbiturates were usually avoided as premedicating drugs.

The anesthetic agents and methods were the same as used on any adult group of patients considering the physical status of the patient and the surgery to be performed.

In the last three years of the series combined anesthesia was used more often. This was especially true of the 70 year old group. When pentothal was used in smaller doses than the usual dose for an adult, it could be safely given to patients over 65 years of age.

The usual anesthetic complications such as laryngospasm, bronchospasm, and arrhythmias were noted. Reflex hypotension was observed and was associated with deep anesthesia on several occasions.

There were two deaths occurring in the operating room.

The operative procedures were corrective so that the patient might survive and live more comfortably.

All deaths which occurred within the first eight days postoperatively were analyzed. The greatest cause of death was of cardio-vascular origin.

CONCLUSIONS

As Eversole[4] so aptly states "It is axiomatic that the safety of all anesthetic agents and methods is in direct proportion to the administrator's familiarity with them. There are specific limitations, dangers, and shortcomings of all agents and methods, but this is true whether the subject is a child, a young adult or a man of 90. The special problem presented by the elderly individual is that since he is more fragile, any complication that is allowed to progress without prompt and effective treatment may be more serious."

Over half of this series received combined anesthesia. When pentothal is not contraindicated it can be used carefully in the elderly patient, combined with nitrous oxide-oxygen mixtures. The use of nitrous oxide-oxygen-ether sequence is still good anesthesia.

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[4] Eversole, Urban H: The Anesthetic Management of the Elderly Patient. S. Clin. North America **34**: 619-626. June, 1954.

Hypotension Following Anesthesia

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In 1950 our attention was first attracted to a shock-like state in post-operative patients which seemed to be connected with anesthesia rather than surgery. These cases indicated neither excessive blood loss nor unusual trauma. The shock-like picture was noteworthy because of the unusually slow pulse rate and because the appearance of the hypotension had been delayed some little time after the anesthetic had been terminated. At first we were at a loss to ascribe a common factor to these cases, but after further study it became apparent that this condition had developed in cases which had quiet and shallow respirations during anesthesia and which had been administered, among other agents, cyclopropane and curare.

In the following cases there seems to be no substantial surgical reason for the picture which developed after the patient had returned from surgery.

Case 1.—A cholecystectomy on a patient induced by pentothal and carried on cyclopropane and curare. The blood pressure in this case was 160/85 and rose gradually at the completion of the operation to 180/85. Shortly after returning from surgery it was noted that this patient was very diaphoretic and pallid. Repeated blood pressure readings averaged 75/50. The pulse was 64.

Case 2.—A cholecystectomy induced with pentothal and carried on cyclopropane, ethylene and curare. At the start of anesthesia the blood pressure was 110/75 and at the conclusion of the operation the blood pressure was 135/80. After returning from surgery, the patient became very diaphoretic and pallid. Repeated blood pressure readings averaged 85/45. The pulse was 76.

Case 3.—A bowel resection, induced with pentothal, carried on cyclopropane and curare. Systolic pressure ranged from 100 at the start to a climax of 130 and was 120 after the operation had been completed. The patient, shortly after surgery, was noted to have a blood pressure of 70/38 and pulse of 60.

Case 4.—A hysterectomy induced with pentothal and carried on cyclopropane and curare,

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whose average systolic pressure during operation was 120, showed symptoms of shock with a blood pressure of 78/58 after surgery and had a pulse of 80.

Case 5. — A cholecystectomy whose systolic pressure was 120 at the beginning of the operation and rose to 150 at the completion. On return from surgery the patient became pallid, perspired profusely, and had repeated blood pressure readings of 70/54 and a pulse of 74.

In all these cases the blood pressure was maintained or raised during anesthesia and did not fall until the patient was separated from the anesthesia machine. The picture was that of ordinary or traumatic shock with the exception of one factor—the very slow pulse rate. None of these cases were fatal and, with treatment, all rallied from their shock in a matter of several hours.

Others have commented on similar experiences. In 1952 Meta described cyclopropane shock. He felt that it is due, at least in part, to a high carbon dioxide level in the blood during anesthesia as a result of the respiratory depressant action of cyclopropane. The blood pressure drops after the stimulating effect of carbon dioxide is removed.

Price, Conner, and Dripps found that the increase in arterial blood pressure observed during cyclopropane administration occurred in the absence of respiratory acidosis. Accumulation of carbon dioxide, in the arterial blood, however, was associated with greater increase in arterial pressures.

At the University of Minnesota Hospitals, Miller and associates studied respiratory acidosis during anesthesia. A portable rapid gas analyzer was used in this study. They demonstrated myocardial toxicity evidenced by ventricular fibrillation when a high alveolar carbon dioxide tension was rapidly converted to a normal one. They were able to demonstrate that 35% of major surgical cases undergoing general anesthesia experience at least a twice normal alveolar carbon dioxide tension during some fifteen minute period.

Fisher has cited figures obtained during experimentation showing the CO_2 content of the alveoli under various conditions. Normally the aleveoli contain 5.6% of CO_2. Under anesthesia and with assisted respirations this rises to 6.8%. If the respirations are not assisted the percentage of CO_2 rises to the alarming figure of 12.3%.

There are few good clinical signs available to tell of carbon dioxide accumulation. Neither cyanosis nor depression of blood oxygen saturation always appears with respiratory acidosis. The Minnesota investigators have noted a critical phase right after the switch over to air in patients with severe respiratory acidosis and they have suggested the possible relationship of this phenomena to certain previously unexplained cases of cardiac arrest. These authors studied the surgical deaths associated with cardiac arrest and they felt that there was a possibility carbon dioxide might be implicated. They concluded that a patient, after prolonged and severe respiratory acidosis, hovers on the brink of disaster if suddenly ventilated with a mixture low in carbon dioxide. They recommended supplementing respiration throughout the period of anesthesia.

In 1952 Maloney and co-workers called attention to the fact that it is very difficult to be sure that there is an adequate tidal volume of gas even if the breathing bag is constantly observed. The very important difference of 100 cc. between an adequate tidal volume of 280 cc. and an inadequate one of 180 cc. may be scarcely noticeable by observing the bag. These men employed a simple ventilation meter to give an adequate measure of respiratory exchange. Even though the anesthetic mixture contains a high percentage of oxygen and may allow oxygenation of the blood, it may be inadequate for the excretion of carbon dioxide. The patient's color may be good while carbon dioxide continues to build up in the blood. These authors conclude from their studies that severe hypo-ventilation occurs much more frequently than is realized.

Watrous, Davis, and Anderson believe that anesthesia deep enough to produce relaxation of abdominal muscles causes at least a degree of respiratory depression. The coincidental depression with corresponding respiratory acidosis warrants the use of assisted respiration to return the alveolar carbon dioxide tension to near normal.

Stormont and associates also observed that the accumulation of carbon dioxide during closed circuit cyclopropane anesthesia can be prevented by assisted respiration.

Whitacre, Fisher, and Wiggin have found that when curare and cyclopropane are used together, assisted respiration is necessary to insure adequate carbon dioxide elimination and oxygen absorption. Assisting or supplementing respiration also helps to correct the depression of the respiratory center to carbon dioxide which occurs with many of the premedication sedatives and many of the anesthetic agents. If respiration is not supplemented, excess carbon dioxide may collect without clinical signs or symptoms. The prevention of this carbon dioxide retention prevents the post-operative reaction known as "cyclopropane shock" which is probably due to the sudden release of an excessive carbon dioxide tension with resulting hypotension.

In studying the effect of the electroencephalogram in the evaluation of carbon dioxide accumulation during surgery, Simeone and associates have noted that, at a given anesthetic level, depression of the brain waves is proportional to the increase in blood content of carbon dioxide.

Gibbs has shown that carbon dioxide accumulation in the blood depresses the potential and frequency of the electroencephalogram.

At the recent Pacific Coast Surgical Association meeting, Tuohy warned of the dangers of curare-like agents producing CO_2 accumulation. At the same meeting, Motley suggested the use of a positive and negative pressure anesthetic machine to prevent such CO_2 accumulations.

Our further observations were made on 124 cases of general anesthesia. In these cases respirations were assisted in an attempt to prevent the accumulation of carbon dioxide. If the gas machine used had a pressure gauge, the breathing bag was compressed until the gauge registered from 5 to 7 mm. of mercury. When using a machine without a pressure gauge, enough pressure was exerted on the bag to make the chest rise substantially. This was done every second, third or fourth respiration, depending upon the amount of depression resulting from the anesthetic agents. In all these cases, cyclopropane was the primary agent, supplemented with curare. The patients ranged in age from 17 to 74 years. The duration of the operation was from 40 minutes to 4 hours and 17 minutes. Fifty-three of these cases lasted from 60-90 minutes, 41 from 1 1/2 hours to 2 hours, and 10 from 2 to 2 1/2 hours. The CO_2 absorption technic was used in all cases.

While hypercapnea developed to some degree in spite of intermittent assisted respiration, it was a great deal less severe than if assisted repirations had not been carried out. Hypotension occurred to some degree in the first few minutes after the patient was separated from the gas machine as the carbon dioxide was exhaled into the atmosphere. If the pressure fell 15 mm. or was as low as 85, treatment was begun by giving 3 to 5 mg. of vasoxyl intravenously. This was repeated in five minutes if the blood pressure had not risen adequately. In all of these 124 cases, when the blood pressure was raised to normal levels in this way it remained stable. No evidence of prolonged post-operative hypotension or secondary shock was noted in any of these cases. Vasoxyl was used because it produces a prompt and prolonged rise in blood pressure without causing central stimulation and, as shown by Dripps, Kester, and Rueben, without causing cardiac irregularities even during cyclopropane anesthesia. Ephedrine and similar vasopressors are definitely contraindicated in association with cyclopropane anesthesia because of cardiac effect.

SUMMARY

1. Hypotension following anesthesia has been studied and the literature reviewed.
2. The rapid dispersion of accumulated CO_2 after termination of anesthesia is thought to be the cause of the hypotension.
3. Complete relief from the syndrome described was obtained by:
   (a) Assisting or supplementing respirations during anesthesia.
   (b) Using vasoxyl postoperatively in some cases as described.

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(Continued on page 67)

Hyperthermia

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In recent months, our attention has been focused on the subjects of hyperthermia, heat retention, heat stroke, and heat pyrexia. These terms apply to high temperatures in individuals who, in our interests, are in the operating room, undergoing, or having undergone, a surgical procedure under the influence of a general anesthetic agent.

It is known that temperatures vary in individuals, due to the loss of control of heat regulating centers of the brain. The patient tends to take on the temperature of his environment, behaving somewhat like a polkilothermal animal. Considerable damage can be done to patients with marked elevation of temperature—convulsions, coma, and psychic disturbances in the recovery stage. It is suggested that liver cells and cells of the central nervous system are the most susceptible to damage by fever. Such damage, determined by autopsy findings, may result in:

(1) General degeneration of the neurons of the central nervous system

(2) Centro-lobular liver necrosis

(3) Lower nephron nephrosis

(4) Wide-spread hemorrhages in the lungs and sub-endocardial tissue

With fever of any kind, there is an increased metabolic demand on oxygen, and if the cardio-respiratory transport system of the body cannot meet the demand, hypoxia of the tissues develops. Pathological lesions, similar to those found in patients dying from the aftermath of fever therapy, are found. Hyperthermia (unusually high fever) places considerable strain on the cardiovascular system. The response of this system to heat decreases the ability of the body to withstand circulatory stress in this way: We all know that the resistance of an individual under anesthesia is lowered, and that there is some degree of traumatic shock in any type of surgery. Handling of tissue with loss of hemoglobin, coupled with hyperthermia, renders the cardiovascular system incapable of performing its function, which is to distribute circulating medium to all parts of the body. With the metabolic demand on oxygen denied, vital organs degenerate and die. The extent of damage depends upon

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the elevation of the temperature and the length of time the fever has existed.

Robert E. Clark, M.D., Louis R. Orkin, M.D., and E. A. Rovenstine, M.D., in their article, "Body Studies in the Anesthetized Man," have brought to light some interesting and helpful facts. A study in body temperatures while patients were under anesthesia was conducted by means of a specially devised machine called a Micromax 6-point recording thermocouple (Leads & Northrop Co., Philadelphia). With the aid of this mechanism, the temperature with the closed system, inside the body, could be determined. The patients selected for surgery were people whose surgical procedure would be from 2 1/2 to 4 1/2 hours duration. The to-fro circle absorption and non-rebreathing technics were used. Cyclopropane and ether were used in the to-fro and circle absorption systems, and nitrous oxide, oxygen, and sodium pentothal for the non-rebreathing system. The patients selected had no history of previous temperature and had been given satisfactory premedication.

The following readings were taken in operating rooms with humidity about 55%:

(1) With temperatures ranging from 90 to 95°, 3 out of 7 patients required treatment for heat retention with ice bag therapy—one after 2 1/2 hours, one after 4 hours, and the other after 4 1/2 hours duration.

(2) With temperatures ranging from 80 to 85°, two out of five developed heat retention symptoms in 3 1/2 hours.

(3) With temperatures ranging from 85 to 89°, three out of sixteen developed symptoms which were apparent at the close of the operation.

All cases of heat retention occured when the to-fro technic was used.

The above studies are sufficient to make us conscious of the presence of conditions promoting heat retention, so that we may endeavor to eliminate such sources that come within our jurisdiction. The principal source of heat gain in the body is the combustion of carbohydrates, fats, and proteins. Add to this operating room lights, warm bodies of the surgeons, hot laparotomy packs and instruments, plus a heavy draping.

Keeping a sterile field is always a consideration of the surgery team. Many thicknesses of drapes for the maintenance of asepsis have been a ruling for years, without too much thought for the patient beneath, regardless of whether there is a rise in the temperature of the room or not. According to the records of the author, the temperature under the drapes was 8 to 10 degrees higher than the room air when temperatures were from 80 to 90° F.

Therefore, with so-called "heat gain" in a patient undergoing surgery, some means of elimination must be attained to keep a balance. Four methods of heat loss were mentioned: (1) Radiation, principally at comfortable room temperatures; (2) Conduction—application of cold towels or icebags, so placed as to cool the body; (3) Convection—warming the air coming in contact with the body, possibly by use of a fan. This method is not recommended in the operating rooms, and therefore is not available to the surgical patient; (4) Evaporation—as the temperature rises, the patient must perspire. Depressing evaporation with the heavy drapings, as in surgery, inhibits the perspiring mechanism, which in turn raises the body temperature. When the perspiring mechanism fails, heat retention is almost certain to develop.

During warm weather, when the room temperature of the surgical theatres is high, the lightest possible draping is advocated. Actually, asepsis is maintained more satisfactorily with light drapes and the patient is less likely to perspire througr them, causing contamination of the field.

Evaporation, by way of the lungs, is another important factor in eliminating "heat gain." It is claimed that an overall 10% of body heat loss is dispersed by this method. The percentage is decreased with the different technics of anesthesia administration. Non-rebreathing systems permit free lung evaporation; yet this is not enough to prevent a rise in body temperature. In the circle absorption system there is less evaporation because of the action of the patient's carbon dioxide on the soda lime. At a lower temperature, the gases return saturated to the patient. There is some condensation in the corrugated tubes and face piece, plus an estimated total heat loss of about 4% of the basal heat production.

With the to-fro system, conditions are different. This system does not permit evaporation of vapor from the lungs. The temperature of this system is hotter than the expired gases. Heat gain is the result, not due to the warm gas, as might be thought, but due to the water condensation in the lungs and airway that it takes to cool the gases to body temperature. The total "heat gain" is about 4% of the basal heat production.

It was felt that the increase in vapor condensation would prove a hazard to the anesthetized patient. It has also been pointed out that in the circle absorption and non-rebreathing systems, condensation does not occur in such amounts. The to-fro system appears to be the one method by which it does. With the rise in temperature of the operating room, the to-fro system may be undesirable from the standpoint of the resulting heat gain. In the experiments mentioned, 9 out of 10 of the heat retention cases were of the to-fro technic. From these facts, I would assume that of the 3 systems, circle absorption and non-rebreathing would be the most favorable methods of administering anesthesia during the warm months and when the surgery is not elective. There is no guarantee that heat retention would not develop, but there are a few precautions that can be taken in the interest of prevention. As mentioned in foregoing paragraphs, with lighter draping many sheets and towels could be eliminated without jeopardizing the surgical technic and sterile field. Wherever possible, during the surgical procedure, frequent lifting of the drapes to permit a circulation of air will aid evaporation. Proper preparation of the patient in regard to fluid balance prior to and during surgery, and replacement of lost fluids in equal amounts will help to eliminate some of the danger. Observation of the patient's increasing warmth, followed by cessation of sweating and a sudden rise in temperature, are urgent indications of heat retention. It has been suggested that readings taken of the posterior nasal temperature would assist in determining the on-coming heat retention.

The treatment progresses with one thought in mind, i.e., a reduction in the temperature as quickly as possible. As stated before, the higher the temperature and the longer its duration, the more danger to liver, central nervous system and other vital organs. Temperature can be reduced by:

(1) Fluids intravenously

(2) Cold towels and ice bags around the chest, axilla, thighs, groin and as far as possible without interfering with the surgery

(3) Ice in the Ether attachment of the machine to cool the inspired air as much as possible

(4) Controlled respirations. It is known that muscular activity increases heat production. Controlled or assisted respirations are associated with less internal heat than respirations that are spontaneous.

(5) Having the patient breathing oxygen, so as to control his own thermoregulatory system.

CASE STUDIES

(1) **Child 11 yrs. Mastoidectomy.** Very toxic with temperature. Anesthesia: Ether urge. Cold application to thighs and chest. Induced under ether and intubated. Carried with ether urge and high oxygen flow. Child kept cold by constant fanning, bathing and undraping, for 2 hrs. and 40 minutes. When temperature rose and became uncontrollable, procedure was discontinued at request of anesthesiologist. Typical case of Hyperthermia because of toxicity and loss of heat control. Patient lived for 4 days postoperative and then expired suddenly. Postmortem showed a cerebral abscess which probably was the main cause of the Hyperthermia.

(2) **Child 5 yrs. Emergency appendectomy.** Toxic with high temperature. Anesthesia: Vinethene to ether urge. Cold packs to chest and upper extremities. Induced with vinethene and carried on ether urge. Not intubated, oral patent airway used. Good exchange throughout. Prepared for surgery. Severe convulsion commenced after skin incision was made. Ether discontinued. Sodium pentothal, 2 1/2% sol^{n}., given intravenously. 6 cc. given before convulsion was controlled. Anesthesia agents changed to cyclopropane 20% and oxygen, supplemented with pentothal, totalling 13 cc. for entire surgical procedure. Child cooled with cold packs, and temperature dropped after surgical drapes had been removed. Convalescence moderately stormy, due to toxicity. Was controlled with antibiotics. Home on 7th day, recovered. Any seri-

ousness of damage done by Hyperthermia possibly was averted by corrective measures.

(3) **Child 4 yrs. Emergency appendectomy.** Toxic with high temperature. Anesthesia: Induced with vinethene and carried with ether urge. Procedure uneventful until the close of the operation. Patient developed severe convulsion. Ether was discontinued and a full flow of oxygen given. Closure was hurried, draping removed, and the child packed in cold towels and ice. Intravenous fluids were given. Temperature did not drop with all measures, rose to 108° R. Child expired suddenly 12 hours postoperatively. This was definitely a case of Hyperthermia due to prolonged toxicity, climaxed by the use of a general anesthetic.

(4) **Child 3 yrs. Hernioplasty.** Elective surgery. Normal temperature prior to surgery. Anesthesia: Vinethene, ether urge. Not intubated. Induced with vinethene and carried with ether urge. Satisfactory respiratory exchange throughout, with patent oral airway. Condition under anesthesia satisfactory until closure was being made. Sudden rise in body temperature. Cold packs around chest and upper extremities applied immediately. Anesthetic discontinued and oxygen given freely. Hurried closure at the request of anesthetist. Temperature when drapes were removed was 100° R., which fell within one hour of convalescence. This child was on the verge of Hyperthermia before controlling measures were used.

(5) **Female 40 yrs. Two procedures:** Cholecystectomy and common duct exploration, and exploration for hemorrhage. Abnormal electrocardiogram of undetermined type. Anesthesia: Sodium pentothal, Bairds sol^n., nitrous oxide and oxygen. Induced with sodium pentothal, intubated and carried on Bairds sol^n. with nitrous oxide and oxygen in a circle absorption technic. Considerable difficulty was encountered in maintaining an adequate oxygen exchange even with an intratracheal tube in place. However, except for some hoarseness, the patient emerged from the anesthetic satisfactorily. Within 12 hours it was evident that there was some internal hemorrhage and that surgical intervention was imperative. During the second surgical procedure, with the same consideration of anesthesia, the patient was slightly cyanotic from induction, and appeared to have a spasm of the bronchial tree throughout the surgical procedure with considerable mucus. Uneventful until closure, when again there was difficulty in maintaining exchange of oxygen. Tracheotomy was recommended and commenced with positive oxygen pressure. Color improved slightly in presence of oxygen. Temperature then rose following surgery and, in spite of measures taken, became uncontrollable. Patient expired on the 9th postoperative day with a temperature of 105°. Anoxia, hemorrhage and shock causing the resulting Hyperthermia—a strain on the cardiovascular system that could not be tolerated in this patient.

(Continued on page 67)

Stress

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By the word "stress" the physicist means the interaction between a force applied to an inanimate object and the resistance to that force offered by the object. The force tends to deform the object and the object resists being deformed. The word "stress" was introduced into medicine to indicate a biologic counterpart of this physical concept. A state of biologic stress occurs in a living body whenever it is affected by an agent capable of doing damage.

This effort of the organism to maintain the *status quo* and the mechanisms utilized by it in so doing have been of high interest to a number of able investigators for many years. A century ago Claude Bernard talked about the *milieu interieur* and the importance of maintaining its constancy in spite of changes in the external environment.

In the 1920's Cannon used the term "homeostasis" to indicate the balance in which the body kept itself in spite of upsetting influences. He worked especially to discover the roles of epinephrine and the autonomic nervous system in this function. He showed, for example, that in a state of emergency demanding effort by the body there is an increase in the amount of epinephrine produced; that this raises the blood pressure, speeds up the heart, decreases the activity of the intestinal tract, dilates the blood vessels going to the skeletal muscles, and, in general, puts the body into readiness for fight or flight, for maximum muscular effort. Epinephrine is the hormone from the adrenal medulla.

A decade later the first potent extracts from the adrenal cortex were called by Hartman, "general tissue hormones," because they seemed necessary for the proper functioning of most all body cells and helped to maintain resistance to infection, to combat fatigue, and to keep the cells hydrated.

A number of investigators described reactions of the body to a variety of agents, as Leriche wrote of the *Maladie postoperatoire*, or post-operation sickness, and the changes in blood chemistry and blood picture which he thought were characteristic of this state. Others reported morphological, functional, and biochemical changes following exposure to certain agents (drugs,

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infections, trauma, burns, cold, etc.). All over the world men were studying shock—its causes, physiology, and treatment.

A different approach, which eventually helped to shed light on the means by which the body marshalled its defenses, was that of Wagner-Jauregg, who, as early as 1887, wrote of the therapeutic effect of fever in the treatment of certain mental diseases, especially general paresis. From his work came the development of fever therapy, foreign protein therapy, and non-specific therapy.

In 1936 Selye first published his observation that the systemic reaction of the body was much the same, regardless of the nature of the damage inflicted. He saw that various stress-producing agents (trauma, toxins, asphyxia, infections, and even nervous strain) produce a syndrome of reaction which consists basically of atrophy of the thymus and lymphoid tissue, gastro-intestinal ulceration, and enlargement of the adrenal cortex. Following the clue of the enlarged adrenal cortex he found that there is, indeed, an increase in the amount of adrenal cortical hormones produced in the state of stress, and that some of the effects of this increased hormone production are of benefit in helping the body adjust to and resist damage. He showed that adrenalectomized animals are very inefficient in adapting themselves unless they are given corticoid hormones. This fits perfectly with what had been known for some time about people suffering from Addison's disease, which is due to failure of function of the adrenal cortex.

A man was brought to the hospital for admission. He arrived in the afternoon and went through the usual admitting procedure, which takes some time, without obvious evidence of being seriously ill; however, he lapsed into coma and died even before he was taken to the ward. Autopsy showed tuberculous destruction of both his adrenals. He had left home early that morning and had had nothing to eat for perhaps eight hours. Knowing how poorly Addisonians maintain blood sugar levels when fasting it seemed likely that this man died of hypoglycemia.

Somewhat similar is the case of a young woman who became irrational and then comatose on the second day of an attack of flu. She was admitted through the Emergency Room. Blood was drawn for examination and then she was given saline and dextrose intravenously. Within a few minutes she regained consciousness. Her blood sugar turned out to be 30 mgms. per 100 cc. That clue led to the theretofore unsuspected diagnosis of Addison's disease.

Another man, known to have Addison's disease, developed acute appendicitis. Fortunately the findings upon physical examination were quite characteristic, because instead of the low-grade fever and the leukocytosis which are part of the picture of acute appendicitis, this man had a temperature of over 40 degrees C. and a W.B.C. of 3400 per cu. mm. These bizarre findings were due to the fact that his body, having no adrenals, could not increase the output of the cortical hormones necessary to put the body into the best position to resist the infection.

Selye showed also that the pituitary gland is essential for the defense reaction. When the pituitary is removed from an animal, he behaves, in many respects, as though adrenalectomized, and displays a reduced resistance to the effects of stressors. This was not unexpected, for it had been known for some time that the adrenal cortex is dependent upon the pituitary for maintenance of its size and function, which are regulated by that pituitary hormone called ACTH, or adrenocorticotrophic hormone.

The natures of the adrenal cortical hormones were made clear when pure preparations of them became available, and it was possible to examine their separate functions. Bit by bit it was established that the adrenal cortex is a very important organ in maintaining the balance of the body in regard to sodium, potassium, water and glucose; that it contributes greatly to the proper functioning of the cardio-vascular, the gastro-intestinal, the urinary, the nervous and the hematopoietic systems. Most recently it has been discovered that it is the organ which, in large part, directs tissue response locally to injury; that is, regulates the amount of inflammatory reaction and connective tissue formation at the site of attack. Probably by way of this tissue control (quite possibly also by means of some other effect) it in some degree determines the resistance of the body to infection.

The hormone which controls sodium, potassium, and water balance is referred to as the "electrolyte-balance" hormone, or the "mineralocorticoid." The name "electrocortin" has recently been suggested for it. It also acts to stimulate the inflammatory and connective tissue response to injury, and in this role it has been referred to as the "prophlogistic" hormone. The growth hormone of the pituitary (STH) affects tissue in the same way, and so cooperates with the prophlogistic hormone of the adrenal. The hormone which has to do with the regulation of the blood sugar, and which, among other things, affects the nervous system and the composition of the blood, is called the "glucocorticoid" or "glucose metabolism" hormone. We believe that hydrocortisone, very similar in its effect to cortisone, is the glucocorticoid produced by the adrenal cortex. This same hormone operates to resist the inflammatory and connective tissue response, and so has been called also the "antiphlogistic" hormone.

As a reaction to injury of whatever nature, it has been fairly well established that the pituitary is stimulated so that it puts out more of the growth hormone (STH), and ACTH. The former acts, in conjunction with the prophlogistic hormone electrocortin, to promote tissue reaction to the damaging agent. This helps in repair and probably also in resistance to infection. The latter, ACTH, stimulates the adrenal to increase its output of hydrocortisone (glucocorticoid and antiphlogistic) which, in turn, acts to increase blood sugar, elevate the white blood cell count, keep fever down, and to resist tissue reaction. All these effects must be in proper balance in order to serve the best interests of the body, and dire results might follow if one should dominate all others. Certainly it has been learned from animal experiment and from observations in humans that with the mineralocorticoid one can produce edema and hypertension, and that with the glucocorticoid one can produce interference with wound healing, diabetes, psychosis, etc. Some recent work suggests that the pathogenesis of some types of leukemia may involve imbalance of the adrenal hormones. In the field of reaction to infection there is much evidence to support this theory. The rat normally is highly resistant to infection with tubercle bacillus; with cortisone or ACTH it may be made quite susceptible.

Recently we had in the hospital a man with functioning adrenal cortical carcinoma with wide-spread metastases, which meant that he had been subjected for a long time to endogenously administered cortical hormone in large doses. He had the usual evidences of Cushing's disease; moon face, buffalo hump, polycythemia, plethora, hypertension, diabetes, osteoporosis, mental changes, and poor resistance to infection. The last of these was dramatically illustrated at autopsy, which disclosed wide-spread infection, including purulent myocarditis.

Consideration of these facts and other pertinent ones will suggest that in them may lie knowledge upon which could be built a new concept of the nature of disease. Certainly we could make use of a fresh approach. Such things as rheumatic fever, rheumatoid arthritis, lupus erythematosus, leukemia, psychosis, gout, most cases of diabetes, hypertension, many skin diseases, allergic reaction, intestinal ulcers, renal disease, and many others as diverse and as important have never been explained in their pathogenesis and, for the most part, have resisted the development of satisfactory therapy. Now it is certain that all of these have some very cogent relationship to the adrenal hormones. Some of them—diabetes, ulcer, hypertension, and psychosis, for example — have been produced in the human by the administration of these hormones; others — gouty attack, asthma, lymphatic leukemia and the rheumatic diseases — have been, at least temporarily, suppressed by them.

Selye has put these things together and has pointed out that, in the process of adaptation to stressor agents, it is possible that there could arise imbalances of one sort or another which in themselves would be capable of producing disease. To diseases arising in this manner he has given the name "diseases of adaptation." Clinically we see diabetes mellitus develop after stress of obesity, hyperthyroidism, or infection. This is especially true in people whose families show a history of the disease. We have for a long time regarded duodenal ulcer as being related in its origin to stress, often emotional, and have known, since Curling's description in 1842, that it sometimes comes into being as the result of burn of the skin.

A lot of ground has been covered in the last quarter of a century. There is no question but that we now have a much clearer perception of the behavior of the body in the state of stress, and we have a good clue to the pathogenesis of many diseases previously considered of unexplainable origin. Today's hope is that we may soon learn how to manipulate treatment so that the balance of the body may be righted and some of these serious diseases may be permanently cured. Further, we hope to learn how to prevent the development of imbalance and disease. Of course this is, and has been, the great aim of Medicine, and actually we have been using some fairly potent tools toward this end for years. Good diet; avoidance of overweight; enough rest; avoidance of injury, extremes of heat and cold, intoxication, and nervous strain all reduce stress and certainly help to minimize the incidence of diseases of adaptation. The whole effort of the anesthetist is to shield the patient from stressors such as pain, nervous shock, anoxia, fatigue, chilling, blood loss, dehydration, and chemical imbalance. It must be remembered that the anesthetic, is, in itself, a stressor of the first magnitude.

Infant Resuscitation After Cesarean Section

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Childbirth by Cesarean Section presents definite problems which make the birth somewhat hazardous. Such a birth is considered an emergency measure. It is an emergency to deliver the child when the mother is malformed or when some other untoward situation, such as abruptio placenta, is evident.

Naturally, the most urgent need is to deliver the child by the safest method and in the shortest possible time. Here is where the anesthetist can do much good by using the technic that will be safest for the mother and for the child.

At Gritman Memorial Hospital in Moscow, Idaho, during the period from September 1951 to September 1954, there were 76 Cesarean Sections. Twenty-eight were done under low spinal anesthesia and 48 under general anesthesia, with no infant or maternal deaths in surgery. Of the 7 successful resuscitations, 1 baby died, due to an esophagotracheal fistula, 24 hours after birth.

GENERAL ANESTHESIA

The technic for general anesthesia is as follows: After the mother is on the operating table, normal saline solution is started intravenously and allowed to drip in slowly. After the child is born, a unit of compatible blood is added to this solution. (Every mother having a Cesarean Section has had her blood typed and crossmatched and has two units of blood in reserve.) The anesthetic is started by letting the mother breathe 100% oxygen for about a minute; then ether is turned on to #1 on the guage. The ether helps stabilize heart action when cyclopropane is given. Then nitrous oxide is started at a flow rate of 150 cc. per minute. The nitrous oxide helps dilute the mixture and prevents the ether and cyclopropane from becoming too irritating. Cyclopropane is increased gradually until the patient can tolerate 600-700 cc. per minute. The mother, however, is the final index as to the tolerance of cyclopropane and the anesthetist must be alert to any sign of the mother's inability to tolerate this concentration.

When the surgeon exposes the uterus, all anesthetics are discontinued, oxygen alone being continued until the child is delivered. After the delivery has been completed, the mother is given pentothal sodium 2 1/2% (Abbott) plus curare (Squibb), nitrous oxide and oxygen.

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LOW SPINAL ANESTHESIA

The low spinal block is administered by the surgeon. The needle is inserted in 3rd lumbar interspace, with the patient in sitting position. After 10 mgm. of pontocaine have been injected into the spinal canal, the patient remains in sitting position for 20 seconds. When the patient is returned to the operating position, an intravenous infusion of normal saline is started. Oxygen is given to the mother to prevent nausea and vomiting. The blood pressure is maintained by regulating the flow of the saline solution and by continuing the administration of oxygen. No stimulants are given to the mother before the birth of the baby.

CARE OF INFANT AFTER DELIVERY

The established procedure for resuscitation has been used with success. Immediately after delivery, the mouth of the child is suctioned with a rubber bulb syringe. If the child does not breathe or cry adequately within two minutes, an infant size laryngoscope blade is used to expose the larynx and vocal cords and a #0 plastic intratracheal tube is inserted into the trachea. A #8 soft rubber catheter is inserted through the intratracheal tube and suction is applied with the Gomco suction machine, the mucus being withdrawn with the catheter. By gently blowing into the intratracheal tube, the lungs are expanded. Oxygen inhalation through the intratracheal tube is given intermittently to simulate the depth and volume of breathing. After the child begins to squirm, raises its arms and legs and has adequate respiratory excursions, the intratracheal tube is withdrawn and oxygen inhalation by mask is instituted. When the child cries and all evidence of cyanosis disappears, he is taken to the nursery. No stimulants are given while the infant is in an anoxic condition. Only after he begins to breathe may one be given.

At Gritman Memorial Hospital each child was resuscitated in the manner described. The children varied in age from 3 weeks premature to term. Six mothers received atropine gr. 1/150 only and the seventh mother had demerol 50 mgm. and atropine gr. 1/150. Four mothers had cyclopropane, nitrous oxide, oxygen and ether for induction. Pentothal sodium, curare, and nitrous oxide-oxygen were used after delivery. Three mothers had low spinal block with pontocaine 10 mgm. for induction and pentothal sodium, nitrous oxide-oxygen postpartum. The condition of the children varied from slight to extreme cyanosis, from active movement to extreme flaccidity, and from full respiratory effort to total absence of respiratory effort. In each child a large amount of mucus was withdrawn by suction and in one case of abruptio placenta a large amount of blood and mucus was withdrawn from the mouth, trachea and bronchi. There was slight crying in two children and total absence of crying in five children. The recovery of six children was complete. One died of a tracheoesophogeal fistula.

It is felt that the children were not under the influence of narcotics due to heavy sedation of the mothers. The average time from induction to severing the umbilical cord was 8-10 minutes, so we feel the anesthetic was not a contributing factor in the depression of the children. In each child, once the mucus was cleared from the mouth, trachea and bronchi, response was immediate.

When it is evident at birth that the child is not going to cry, resuscitation procedures should be started immediately "since permanent brain damage due to oxygen want is swift and devastating."[1] It is my belief that the incidence of brain damage is in inverse ratio to the speed with which artificial resuscitation is instituted.

Livingstone has said, "The object of resuscitation is to remove carbon dioxide from the alveoli and introduce oxygen."[1] Giving carbon dioxide-oxygen mixture to stimulate respiration is a common fallacy. Since there is an abundant supply of carbon dioxide in the body, adding more carbon dioxide will not stimulate the child to breathe, but actually will further depress the respiration.

Kreiselman[2] has succinctly listed the causes of neonatal apnea: "The failure of the child to breathe may be due to asphyxiation, immaturity, brain damage and drugs." I believe that some of the causes of these factors may be:

1. The lack of immediate suctioning to remove mucus from the child's mouth. (asphyxia)
2. Lack of speed on the part of the surgeon. The longer the mother is under anesthesia before delivery, the greater the amount of anesthetic in the blood stream of the mother. (drugs)
3. The umbilical cord being wrapped around the neck of the child, lessening the supply of oxygen. (asphyxia)
4. Mal-position of child in intrauterine life. (asphyxia and/or brain damage)
5. Improper diet of the mother. This may cause the child to become weakened through mal-nourishment. (immaturity)

Conclusions

Every anesthetist should be able to do infant intratracheal intubation and to suction the trachea and bronchi instead of waiting until an irreversible condition has developed.

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Livingstone, H. M.: Safety measures in oxygen therapy, Hosp. Management **70**:42-43, July 1950; **70**:36-38, Aug. 1950; **70**:46-48, Sept. 1950.

Kreiselman, J.: An improved apparatus for treating asphyxia of the newborn infant, Am. J. Obst. & Gynec. **39**:888-890, May 1940.

Notes and Case Reports

PENTOTHAL SODIUM BY RECTUM

Pentothal sodium given rectally for preanesthetic hypnosis, or, if desired, for basal anesthesia, is rapidly assuming an important position in the armamentarium of the modern anesthetist. Here at Lake City General Hospital in Couer d'Álene, Idaho, it quickly became more popular than the slow acting barbiturate suppositories for pediatric premedication. This was due mainly to its ability to produce a rapid, smooth somnolence before the child is removed from his room, sparing him the subsequent terrors of the unfamiliar operating room. The profound reduction in parental anxiety is in itself enough to justify its use.

Initially, we found that a 10% solution of rectal pentothal in tap water was somewhat irritating to the rectal mucosa and frequently was the cause of an involuntary stool shortly after the child drifted off to sleep. This problem was solved by replacing the tap water with a thick, boiled corn starch solution. The dry drug is added to the starch water which is so concentrated that it is drawn into a syringe with difficulty. The method is as follows: Place the amount of water needed for a stock solution in a pan and heat until it comes to a boil. Add cold water to the starch powder and stir until a thick paste is formed. Add this mixture to the boiling water and allow it to boil until it becomes translucent. Allow the starch water to cool until it has reached room temperature, then add the required amount of pentothal to get a 10% solution. Make certain that the dry drug is completely dissolved in the starch water.

Perhaps rectal pentothal plays its greatest role in the pediatric emergency. For children who have sustained simple fractures, we have simply instilled the required amount of rectal pentothal for somnolence, supplemented it with nitrous oxide and oxygen, and have found this to be entirely adequate. The fact that this combination is non-explosive assumes great importance when one remembers that many emergencies are handled in the X-ray room. Nausea and vomiting are conspicuous by their absence.

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Legislation

Emanuel Hayt, LLB., Counsel, A.A.N.A.

Injury To Student Nurse Held To Be Subject To Workmen's Compensation Law—The Highland Hospital moved to dismiss the complaint against it on the ground that the Court did not have jurisdiction of the action. The plaintiff, father and administrator of deceased, alleged in the first cause of action of his complaint that the decedent became a student nurse in the employ of the defendant Hospital in September 1950 and continued as such until her death on July 6, 1951, that part of defendant Hospital's agreement with the decedent was that it would supply doctors for any illness that she should incur as a student, that the defendant Hospital negligently assigned the decedent to contagious disease cases, and failed to use due care in its supervision of decedent and in its selection of physicians and instructors it employed and in selecting and supplying equipment used in the practice of nursing, and failed to give proper instruction for sterilizing and preparing instruments used in connection with the treatment of contagious diseases.

The court held as follows:

"It being undisputed that the decedent was covered by workmen's compensation and, as alleged in the first and fourth causes of action, that decedent was employed as a student nurse at the time of her illness and death and was treated in the course of her employment, plaintiff's exclusive remedy in said causes of action is with the Workmen's Compensation Board.

"The second cause of action stands on a somewhat different footing. Although it is therein alleged that decedent was a student nurse in training with the defendant Hospital, it is not alleged that she was treated in the course of her employment, bût it is alleged that she entered the hospital as a patient for treatment and that through the negligence of defendant Hospital in employing its physicians and nurses and through their negligence in treating decedent, she died.

"The motion herein is directed to the Court's lack of jurisdiction of the subject matter. Clearly, the Court has jurisdiction of this subject matter, assuming that there was no relationship between decedent's employment and her illness and death. But it is equally clear that the above facts do not constitute a valid cause of action against the defendant Hospital since there is no allegation of administrative negligence on the part of the Hospital. The scant references in the complaint to faulty equipment, made in context as they are with the theory

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of defendant Hospital's negligence through its physicians and nurses, cannot support a cause of action for administrative negligence on the part of the Hospital.

"Under said defendant's request for other relief, it seems proper, therefore, to rule on this cause of action as a matter of law. Hence all three of said causes of action must be dismissed.

"The plaintiff indicates in his opposing affidavits that he now seeks to stand on a different set of facts from those originally pleaded. An amended complaint may contain a cause of action that is not subject to the motion made herein. The motion is therefore granted in favor of the defendant Hospital with leave to the plaintiff to serve an amended complaint within twenty days of the service of a copy of the order to be entered hereon with notice of entry."

(Glaser v. Highland Hospital, 131 N. Y. S. 2d 728)

Appeals Court Affirms Dismissal Of Gold Therapy Case Against Dermatologists—This action was brought on the theory that defendant doctors proximately caused the jaundice by carelessly and negligently administering excessive amounts of heavy metal salts, over the protest of plaintiff, after toxic symptoms had developed. It was also charged that defendants were negligent in treating plaintiff without advising him of the risk and danger incident to the treatment.

The parties agreed that the only issue raised on this appeal was whether defendant doctors were negligent in the administration of gold injections.

Plaintiff consulted defendant dermatologists, Pommerening and Shaw, concerning skin lesions on his face and neck. Dr. Pommerening diagnosed the skin lesions as chronic discoid lupus erythematosus. Dr. Shaw confirmed the diagnosis.

The injection of heavy metal is a recognized treatment for this disease. The therapy consists of periodic injections of heavy metal salts for a definite time, until an effective amount of the therapeutic agent has been administered. Continuity of injections is necessary for the success of the treatment.

The medical testimony disclosed that gold therapy was the usual and standard treatment, in the community, for chronic discoid lupus erythematosus; that defendant doctors used a proper and recognized dosage; that continuity of injections was important for success in the treatment; that it was not the customary standard of practice to tell the patient all the risks involved nor to recite the symptoms; that the judgment of the individual doctor had to be exercised in the light of the mental and psychosomatic make-up of the patient in advising of the risk involved; that when possible symptoms of toxicity arose, they had to be evaluated in relation to the progress gained in the therapy in making a decision whether to continue or stop the treatment.

The trial court found: that Dr. Pommerening had made the usual and customary examination and diagnosis of plaintiff's skin disease; that the treatments of heavy metal therapy, the dosages, the intervals in which the injections were made, and the information given or not given to plaintiff, all represented the usual, customary, and standard practice of dermatologists in the community; that defendant doctors did not deviate from the usual standard practice of dermatologists in the community; and that they were not negligent in treating plaintiff as they did, including the last gold treatment.

The judgment of dismissal was affirmed.

(Woods v. Pommerening et al., 3 **CCH Neg. Cases** 2d 1022—Wash.)

Expert Testimony Lacking To Prove Malpractice In Diathermy Treatment—The plaintiff offered evidence to prove the following facts: The defendant is a physician engaged in the general practice of medicine in Naugatuck. On August 28, 1949, the plaintiff sprained his right wrist and on the next day went to the defendant about the injury. After examining the wrist, the defendant first massaged it and then applied an ace bandage. The plaintiff returned to the defendant's office on September 3, 1949. On that occasion, the defendant decided to apply special treatment to the wrist. For that purpose he took the plaintiff to a room located in the rear of the large house which the defendant used both as his offices and as his home. In this room, which was about fifteen feet from his private office, was a diathermy machine. After having his patient sit down on a stool, the defendant put around the plaintiff's wrist and forearm rubber cuffs which were connected by cables to the machine. The defendant did not apply any padding or gauze to the plaintiff's skin where it was in contact with the cuffs. After first turning some dials on the machine, the defendant switched it on. He then left the room without giving the plaintiff any instructions as to the cuffs or the machine.

About fifteen minutes later, the plaintiff, feeling a burning sensation in his wrist, called loudly for the defendant but received no response. The plaintiff thereafter continued to call but it was not until another fifteen minutes had passed that the defendant came to the treatment room and turned the machine off. Upon removal of the cuffs the plaintiff's wrist was found to be reddened. Subsequently the wrist became blistered, and although treated by the defendant until February, 1950, it failed to heal properly. The burn has left a permanent scar which may require surgery, and the strength of the hand has lessened.

Upon the trial the jury returned a defendant's verdict, from which plaintiff appealed on the ground that the court refused to charge the jury in a particular requested by him. The request ran to the effect that if the diathermy machine was equipped with an alarm signal which could be set to give the patient warning of the end of the treatment and if "the exercise of reasonable care, skill and diligence required, that such alarm signal should be set and [if] the defendant failed to set said alarm signal then you may find the defendant liable for malpractice."

In the ordinary action for negligence, the court held, the jury requires no evidence as to the degree of care a defendant should use under the circumstances. This does not apply, however, to an action for malpractice. Since the members of the jury are laymen, they cannot be expected to know the requirements for proper medical treaament in the usual case. The evidence of experts is ordinarily necessary. The only exception is to be found in those instances where there is manifest such an obviously gross want of care or skill as to afford, of itself, an almost conclusive inference of lack of care or skill and thus to dispense with the necessity of testimony by expert witnesses.

"The case at bar does not fall within the exception. Since the plaintiff failed to offer any expert evidence on the subject, the court was warranted in refusing to submit the request to the jury."

(Marchlewski v. Cassella, 3 CCH Neg. Cases 2d 1039—Conn.)

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Book Reviews

GYNECOLOGY AND GYNECOLOGIC NURSING. By Norman F. Miller, M.D., Prof. of Obstetrics and Gynecology, University of Michigan Medical School; and Hazel Avery, A.B., R.N., Ass't. Professor of Nursing, University of Michigan Hospital; Supervisor of Nursing, Women's Hospital, University of Michigan. Cloth. 3rd edition 525 pages. Philadelphia and London: W. B. Saunders Co., 1954.

To anesthetists who are working with patients who have gynecologic problems, this book will be useful in many ways. The bases for many of the conditions that bring women to surgery will be of value in better understanding of the patient. Special factors of position, body mechanics, pathology and operative procedure, as presented in this book, will make the nurse anesthetist a more understanding and valuable member of the gynecologic surgical team.

FIRST AID AND RESUSCITATION. Carl B. Young, Jr., M.P.H., Instructor, Emergency First Aid; Former Ass't. Director, Harris County Emergency Corps, Inc., Houston, Texas; Member, International Rescue and First Aid Association; First Lieutenant, Medical Service Corps, U.S.A.R. 338 pages, 169 figures. Charles C Thomas, Springfield, Ill., 1954. $8.50.

Written by a man who has had first-hand experience in rescue, first aid and resuscitation, this book is meant for persons who may appear on the scene of an accident before a physician arrives. Believing that a person who understands the reason for certain first-aid measures will do more good than one who follows rules that tell only the "how" and not the "why", the author has included more than the usual do's and don'ts that appear in first-aid manuals. Liberally illustrated with many gruesome pictures taken at the scenes of accidents, suicides and murders, the book is impressive for more than its text. Anesthetists will find many useful suggestions for application, not only in first-aid, but in managing injured persons who require anesthesia.

NURSING. Cecilia L. Schulz, R.N., Babylon, New York. Vocational and Professional Monographs, papers, 20 pages. Boston, Mass.: Bellman Publishing Co., 1946. $1.00.

This small, paper-covered monograph is one of a series designed for use in vocational and professional guidance. It has presented the subject of nursing from an historical, a practical and an inspirational point of view. Advice to the prospective nurse includes methods of judging personal fitness for the profession, methods of evaluating schools of nursing, and job opportunities for the nurse. By using 1938 figures in presenting the salary scales, the author seems to have unfairly jeopardized the otherwise stimulating presentation.

A Textbook of Surgery For Nurses. Edward S. Stafford, B.A., M.D., F.A. C.S., Associate Prof. of Surgery, Johns Hopkins Univ.; Surgeon, Johns Hopkins Hospital; Lecturer in General Surgery, Johns Hopkins Hospital School of Nursing; and Doris Diller, B.A., R.N., Associate Prof. of Nursing and Director of the Cancer Control Project, Skidmore College Dept. of Nursing, New York City; formerly Instructor and Supervisor, Surgical Nursing, Johns Hopkins Hospital School of Nursing. 2nd ed., illustrated. Philadelphia and London: W. B. Saunders Co., 1954.

This second edition of a nursing textbook includes information that will be found useful to anesthetists as a frame of reference for instructing others in the care of patients who have been or are about to be anesthetized. A chapter on the postoperative care of patients includes the procedures, equipment and problems that a nurse needs to know when working in a recovery room. Special problems of the airway, shock and positioning of the patients will be found of interest to all anesthetists who are responsible for the pre- and post-anesthesia care of the patient.

Psychology and the Nurse. By Frank J. O'Hara, C.S.C., Ph. D., Dean of the Science Division, King's College; Professor of Psychology, Mercy Hospital School of Nursing, Wilkes-Barre, Pa. Cloth. New, 4th ed. 313 pages with 23 figures. Philadelphia and London: W.B. Saunders Co., 1954. $3.50.

This fourth edition of a well-known book on psychology in relation to nursing continues the original plan, presenting the subject in a manner that will be understandable to beginning student nurses. Certain portions of the book have been brought up to date and reflect the most recent thinking on the subject. Each chapter is followed by a series of questions for review and topics for classroom discussion as well as resource material. This book should be of interest to anesthetists who have need for knowledge of the latest developments in psychology in relation to nursing.

The Art, Science and Spirit of Nursing. Alice L. Price, R.N., M.A., Counselor, School of Nursing, Presbyterian Hospital, Chicago. Cloth. 882 pages with 275 figures. Philadelphia and London: W. B. Saunders Co., 1954. $5.50.

In this book the author has followed a plan of presenting the subjects in an order similar to that in which they may present themselves to a nurse during her training. Beginning with the new student, a chapter is devoted to an indoctrination period, later to the housekeeping in hospitals and thus on to the increasingly complex steps in nursing care. Pictures from many hospitals add to the interest of the text. Each chapter is preceded by a topical outline and is followed by references for reading. At the beginning of each chapter, a cartoon shows the humorous side of nursing. These may make the book palatable to the reluctant reader, but this reviewer believes that they are not appropriate in this type of text. It is hoped that no relative of a dying patient sees the cartoon that heads the chapter, "Care of the Dying Patient", in which a patient speaking to a nurse and a doctor says, "If I can't take it with me, I ain't goin'."

ELEMENTS OF PEDIATRIC ANESTHESIA. C.R. Stephen, B. Sc., M.D.C.M., F.A. C.A.D.A. (R.C.P. & S.) Prof. of Anesthesiology, Duke Univ.; Chief, Div. of Anesthesia, Duke Univ. Hospital, Durham, N.C.; Formerly, Chief, Dept. of Anesthesia, Children's Memorial Hospital, Montreal, Quebec, Canada. 109 pages. Charles C Thomas, Springfield, Ill., 1954. $3.50.

To every anesthetist seeking information on the complex and often bewildering problems of anesthesia for children, this book will be most welcome. It is difficult to believe that so much of value can be condensed into so small a volume. The size actually lends merit to this monograph for it encourages an incisiveness that enhances its value. Placing much emphasis on the physiologic basis for special technics for pediatric anesthesia, the author has laid a foundation for understanding the later chapters on premedication, technics and drugs. Special surgical procedures are presented in brief manner that, read out of context, would seem to be too cursory. However, reading the book as a whole, this does not prove to be true.

FUNDAMENTALS OF ANESTHESIOLOGY FOR THE NURSE ANESTHETIST. The notes that are included in the course of anesthesia for nurses at Sacred Heart Hospital, Allentown, Pennsylvania, have been collected by the Director, Sr. M. Bonosa. They have been mimeographed and bound in russet fiberboard and are available for sale to members of A.A.N.A. Included in the notes are excerpts from many sources, references for each division, outline drawings and charts. The notes have been indexed. 482 pages. $7.00.

STELLATE GANGLION BLOCK. Daniel C. Moore, M.D., Director, Dept. of Anesthesiology, Mason Clinic; Chief of Anesthesia, Virginia Mason Hospital, Seattle, Wash. Cloth. 280 pages. Charles C Thomas, Springfield, Ill., 1954. $10.50.

This book presents the many facets of a relatively new and special technic of regional anesthesia. Profiting from the greater experience of the French, the author has consulted the French literature and has added to it a review of the English language literature as well as an extensive experience in this method. In addition to the carefully prepared anatomic drawings and photographs, much care has been used in presenting every phase of this complex blocking procedure. Special conditions that have been treated with stellate ganglion block are presented in separate chapters of the book.

The TWENTY-FIRST QUALIFYING EXAMINATION for membership in the American Association of Nurse Anesthetists will be conducted on Saturday, May 14, 1955.

The deadline for accepting completed applications, including the transcripts, is April 4. If application without transcript is received too close to the deadline, the application may not be considered by the committee in time for the candidate to be scheduled for the May examination. Notice of eligibility will be mailed about April 10.

Abstracts

Dobkin, A. B. and Gilbert, R. G. B.: Physiological effects of chlorpromazine. Anaesthesia. **9**:157-174 (July) 1954.

"In the Spring of 1953, clinical trial in several Montreal hospitals of a new phenothiazine derivative . . .known as Largactil, Thorazine RP4560, M & B. 2378 and 2601A SKF. . . . This paper summarizes the results of investigations undertaken to aid the cautious introduction of this drug of multiple actions. Each of these actions were studies on a controlled and individual basis. . . . The overall effect of this drug indicates usefulness in poor risk patients who require very cautious management with anesthetic agents for urgent surgical procedures. This drug is valuable as a premedicant when used alone for cases undergoing surgery with spinal, regional or local analgesia. For general anesthesia, a therapeutic dose of atropine or a small dose of demerol (pethidine) (10 mg.) is indicated. . . . This drug evidently induces general mild depression of all vital organs (brain, heart, lung, liver) when given in therapeutic doses. This effect is additive to all forms of narcotics, relaxants and anesthetic drugs. . . .

"Doses exceeding 150 mg. per day by mouth for long periods may cause liver dysfunction. Doses exceeding 100 mg. per day intramuscularly may cause myocardial depression. It is unsafe to administer doses exceeding 25 mg. intravenously without the precautions accorded administration of other cardio-vascular and respiratory depressants. This drug induces complete sympathetic nervous system depression lasting 4-6 hours. The peripheral vasodilatation induced indicates usefulness as a diagnostic and therapeutic agent in peripheral and cerebral vascular diseases. Hypothermia and hypotension are facilitated under these circumstances. The antiemetic effect of this drug is truly remarkable. Suppression of salivary and gastric secretion is evident, but the mechanism of this action is not clear. The use of this drug in traumatic shock should await more conclusive physiological evidence that peripheral vasodilatation is desirable in such circumstances. This drug should not be used in the presence of severe depression of the vital organs (cerebral, cardiac, pulmonary or hepatic)."

Dundee, J. W.; Mesham, P. R.; and Scott, W. E. B.: Chlorpromazine and the production of hypothermia. Anaesthesia **9**:296-302 (Oct.) 1954.

"In recent months considerable interest has been aroused by the use of 'artificial hibernation' and induced hypothermia in major surgical operations. These technics often involve the use of the drug chlorpromazine. . . . The present paper is an endeavor to determine in some part which of the many alleged properties of chlorpromazine play a part in its thermolytic action. In addition, a comparison has been made between the efficiency of chlorpromazine in lowering body temperature with that of a commonly used anti-pyretic drug. . . .

"Experiment 1—Effect of various drugs and combinations of drugs on the average fall in rectal temperature, and on the amount of shivering in the dog after covering with ice. . . . Experiment 2—Effect of various drugs and combinations of drugs on established shivering. . . . Experiment 3—The effect of chlorpromazine and hexamethonium bromide with procaine amide on the body temperature of rats exposed to a low environmental temperature. . . . Adrenolytic agents or ganglion blocking drugs have no effect on shivering although because of vasodilatation they may slightly aid the production of hypothermia. Large doses of morphia, while inhibiting shivering, do not appreciably affect the rate of fall in rectal temperature. Of the drugs studied, chlorpromazine alone possesses the ability to produce vasodilatation and to inhibit shivering, and proved to be the most effective in aiding the production of hypothermia."

Reckless, D.: Potentiation. Anaesthesia **9**:288-295 (Oct.) 1954.

"Although many drugs are synergistic, chlorpromazine (Largactil) and promethazine (phenergan) exhibit this property par excellence. To demonstrate their activity, the spirometer tracing is an excellent method, recording variations in the respiratory pattern which would otherwise escape clinical observation. . . . The effect of these drugs on thiopentone is also easily recognized, although pethidine would appear to play a part in its potentiation . . . . The amount of thiopentone used and the ensuing supplementary anesthesia is inversely proportional to the amount of pethidine in the mixture. . . . Ether is undoubtedly potentiated by chlorpromazine and promethazine . . . . Chlorpromazine and promethazine produce no effect on curarised respiration, where the anesthetic is nitrous oxide-oxygen.

"When hypothermic-ether-tubarine sequence is used for upper abdominal surgery, the summation produces excellent relaxation in the presence of adequate respiratory excursion. After the closure of the peritoneum, it is not ether which is eliminated but potentiated ether. The need for reversal of residual myoneural block by neostigmine is not required. But where nitrous oxide-oxygen and trilene is used to supplement hypothermia, and also in ordinary anesthesias for upper abdominal surgery, small doses of neostigmine are necessary in 75% of cases. . . . Chlorpromazine and to a lesser degree promethazine, have a marked potentiating action on pethidine and morphine analgesia. It might be assumed that the respiratory depressant effects of these latter drugs would also be accentuated. Yet, if sufficient pethidine or morphia is given to cause respiratory arrest, chlorpromazine exerts a paradoxical effect; for respiration is restarted. . . . In this respect, promethazine is unreliable. . . . With large doses of pethidine or morphia, no supplementary anesthesia is required at all, but it is necessary to potentiate the analgesia of these drugs by hypothermia."

Sadove, M. S.; Levin, M. J.; Rose, R. F.; Schwartz, Lester; and Witt, F. W.: Chlorpromazine and narcotics in the management of pain of malignant lesions. J.A.M.A. **155**:626-628 (June) 1954.

"Experimental work by Courvosier and Fournel has shown that a new phenothiazine compound chlorpromazine (a French generic name for 10-y-dimethylaminopropyl -2-chlorophenothiazine hydrochloride) intensifies and prolongs the action of various drugs, notably narcotics, hypnotics, anesthetics, and muscle relaxants. . . . Chemically, chlorpromazine is. . . .structurally related to the antihistaminic Phenergan. . . .and to the antispasmodic Diparcol. . . . Patients with chronic, severe pain inadequately relieved by large or increasing doses of narcotics were selected for study. A total of 30 patients were observed. All had severe pain, and all but one had cancer. . . . Fourteen of 18 patients, when given chlorpromazine, obtained satisfactory analgesia with dosages of narcotics that had previously been inadequate for relieving their pains; one obtained fair relief; one reported no relief. The two other patients defaulted in taking medication. One. . . .did not take chlorpromazine after the first day, because he believed the drug caused bad dreams and disturbed his sleep; the other. . . .in whom no adverse effects occurred, left the hospital after one week and refused to continue treatment. . .

"Our results indicate that chlorpromazine given with narcotics significantly reduces the patient's requirements for narcotics and provides equal or better analgesia than high dosages of narcotics alone. This enhanced analgesia is, in part, related to the ability of chlorpromazine to alter the patient's reaction to pain. We observed that patients, some of whom stated that the degree of their pain was not appreciably altered, were more relaxed and had a more cheerful outlook. Patients with nausea and vomiting secondary to disease greatly benefited from taking the drug. Since nausea and vomiting are frequent symptoms in patients with advanced malignant lesions, chlorpromazine thus serves two purposes here."

Hershenson, B. B., Isaac, S. J., Romney, S. L. and Reid, D. E.: A new sedative (antipsychomotor) drug useful in labor; preliminary report. New England J. Med. **251**:216-219 (Aug. 5) 1954.

"We recently had the opportunity to observe the antiemetic effects of a new synthetic drug, chlorpromazine. . . . A chance observation in a patient who had annoying vomiting in labor suggested to one of us that the drug might have specific merit as a pharmacologic antagonist to the psychomotor hyperactivity induced by the barbiturates and scopolamine. . . . This communication is regarded as a preliminary report on the use of the drug in labor, for it involves 100 cases. . . . The drug is known in the United States as compound SKF 2601-A, chlorpromazine and Thorazine. . . . We employed a 2.5 per cent aqueous solution (25 mg. per cubic centimeter) with a pH value of approximately 4.9 . . . . The new preparation. . . . has distinct value for the mother during labor, and in this series no ill effects on the fetus were encountered. Its value appears to be as a pharmacologic antagonist to psychomotor hyperactivity periodically encountered in the use of scopolamine in labor. A recommended regimen of medication during labor employing a barbiturate as a psychic sedative, scopolamine as an amnesic agent and chlorpromazine as a psychomotor antagonist is considered. The quieting effect on the patient is impressive. There is much to be learned about the clinical pharmacologic effects of chlorpromazine and its proper dosage. Further investigation of the application of this new drug to obstetric patients is recommended."

Moyer, J. H., Kent, Bartis, Knight, R. W., Morris, George, Dizon, Miguel, Rogers, Stanley and Spurr, Charles: Clinical studies of an anti-emetic agent, chlorpromazine. Am. J. Med. Sc. **228**: 174-189 (Aug.) 1954.

"In this study chlorpromazine (SKF-2601A) was evaluated (clinically) as to its therapeutic potentialities as an anti-emetic agent. Three hundred and six patients were selected in whom the diagnosis and cause of the vomiting had been reasonably well established and in whom vomiting was a definite therapeutic problem. . . . Chlorpromazine is an effective agent for blocking the mechanism of nausea and vomiting without prohibitive side effects. However, before the drug is used the cause of the nausea and vomiting should be reasonably well established. Otherwise, serious clinical conditions may be obscured because of the wide spectra of effectiveness of this agent. There is no evidence thus far to indicate that the drug has any dangerous untoward effects, although, one out of over 500 patients who have received the drug developed jaundice without laboratory evidence of hepatocellular damage. The initial doses are frequently associated with dizziness and sometimes may cause orthostatic hypotension . . . . Should dizziness occur when the patient is upright, it is easily controlled by having the patient remain supine for a short period of time."

Morris, George; Mathews, William and Meyer, John: Clinical experience with chlorpromazine in spinal anesthesia. Anesth. and Analg. **33**:340-343 (Sept.-Oct.) 1954.

"Chlorpromazine (a phenothiazine derivative) is an effective antiemetic agent which acts centrally on the vomiting center and depresses the vomiting reflex. It inhibits nausea and vomiting brought on by a variety of etiologic factors. Although chemically related to the antihistaminic phenergan, chlorpromazine has little, if any, antihistaminic activity: phenergan, on the other hand, has no appreciable antiemetic action. From previous clinical experience, we felt that chlorpromazine might prove useful for inhibiting nausea and retching frequently encountered during spinal anesthesia; therefore, we undertook this study to determine whether this was so. In this report we relate our experience with chlorpromazine in two groups of surgical patients. The first group consisted of twenty-three consecutive patients who were given chlorpromazine for nausea and retching encountered during spinal anesthesia. The second group consisted of seven cases in whom nausea and retching were purposefully induced by gastric traction. Each of these seven was stimulated twice; once, before chlorpromazine was given; once, afterward. . . .

"Nausea and retching incident to spinal anesthesia were alleviated by the drug in 28 of the patients treated. The side reactions of drowsiness and potentiation of premedication were not considered undesirable."

Arner, Ored: Complications following spinal anesthesia. Their significance and a technic to reduce their incidence. Acta Chir. Scandinav. Supp. **167** 1-146, 1952.

"Spinal anesthesia is a method which, by virtue of the relatively simple technical procedure and the excellent analgesia and muscular relaxation, presents major advantages. In recent years, however, very severe criticism has been levelled at spinal anesthesia, particularly following investigations in Scandinavia, on account of both the complications that are considered to occur sometimes during anesthesia, and the late complications, especially neurologic ones of various types .... The present investigation was designed to elucidate the incidence and significance of various complications that may attend or follow spinal anesthesia, and whether the incidence can be reduced by observing a meticulous technic and by selecting the patients so that the method may be used only on specific indications. The investigation seems to have shown that: (1) Spinal anesthesia as well as other anesthetic methods may be followed by complications, and neither spinal nor any other type of anesthesia can be regarded as the ideal, fully risk-free method suitable for use in all situations. (2) Careful preoperative evaluation of the patients, the establishment of indications based thereon, as well as meticulous sterile precautions, accurate technic, and supervision of the patients during operation are of primary importance if the incidence of complications attending this anesthetic method is to be reduced. (3) Both complications during anesthesia and postoperative complications are dependent on the nature of the operation, and only a minor proportion of them are due to the anesthesia itself. (4) The postoperative course after abdominal operations of similar type performed under spinal, ether or intravenous anesthesia is—at all events with regard to good risks—approximately the same, taking into account all the different complications, regardless of the anesthetic method. (5) The principal complication that may be attributed to the anesthesia in operations performed under spinal anesthesia is post-lumbar puncture headache. This headache is seldom protracted and, as a rule, has no effect on the course of the disease as a whole. Other neurologic complications seem to be extremely rare with the spinal anesthesia technic described by the writer. (6) The present investigation enabled the writer to evaluate follow-up investigations conducted by questionnaire. It led to the definite conviction that follow-up investigations based wholly or in great part on replies to questionnaires are of very limited value and may give rise to completely misleading results. The views which have been held in recent years regarding complications of spinal anesthesia are largely based on such investigations.

"The present results suggest that the opinion expressed by some authors during the past few years—namely, that the complications following spinal anesthesia are both serious and common—cannot be correct. Neither the complications during nor those after spinal anesthesia are of such nature and incidence as to justify a further restriction in the use of that anesthetic method. Spinal anesthesia, if used correctly, after due critical appraisal and on specific indications, is a method of very great value in modern surgical anesthesia."

HYPERTHERMIA

(Continued from page 44)

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Control of Hemorrhage

(Continued from page 17)

the effectiveness and safety of such technics will depend upon the understanding and application of the basic principles involved.

In the final analysis, the successful use of any of these measures requires the ingenious planning and performance of both anesthetist and surgeon working in close collaboration. Let us remember that we best serve the traditions of each of our respective fields of specialty by complete unity of purpose—the welfare of the patient!

HYPOTENSION

(Continued from page 39)

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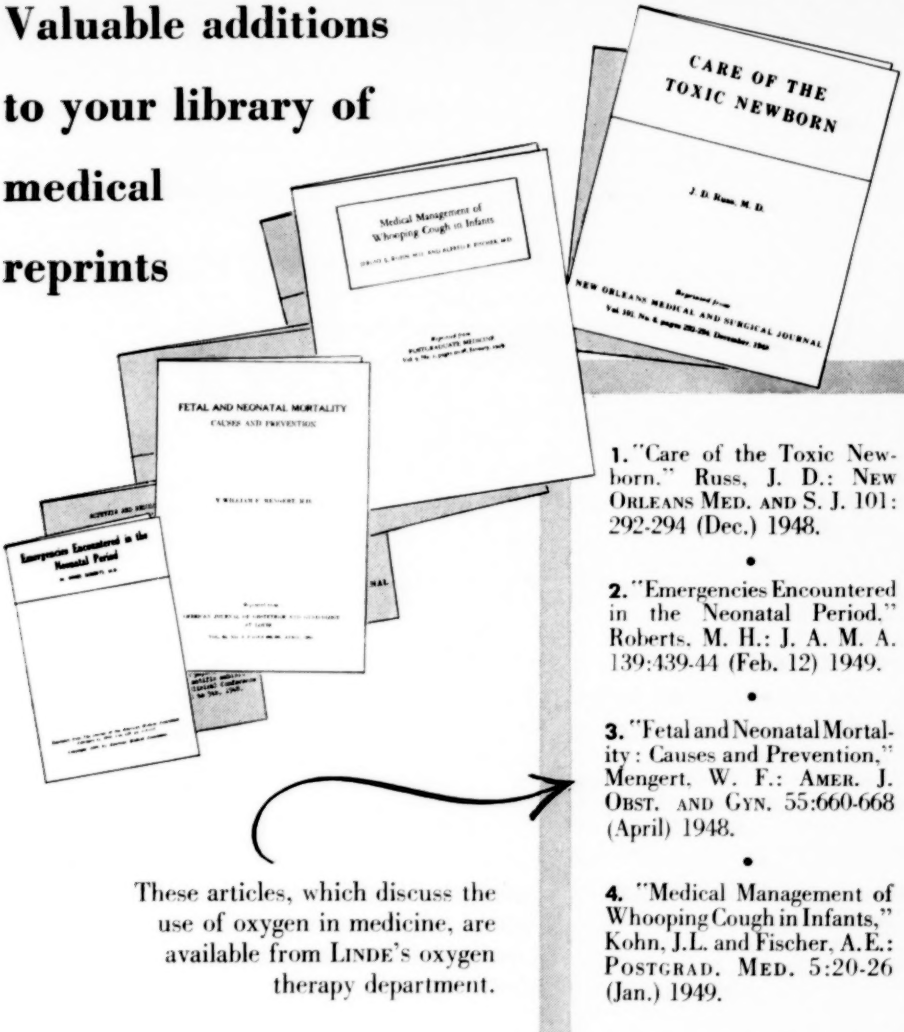

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